DR. WALTER E. DANDY BIRTHDAY NUMBER

Vol. 19 MAY, 1946 No. 5

SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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Vol. 19, No. 5, May, 1946, Surgery is published monthly by The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, Mo. Subscription price: United States, its possessions, Pan-American Countries, $10.00 a year; Canada, $11.50 (Canadian Currency); Foreign, $11.00. Entered as Second-Class Matter January 20, 1937, at the Post Office at St. Louis, Missouri, under the Act of March 3, 1879. Printed in U. S. A.

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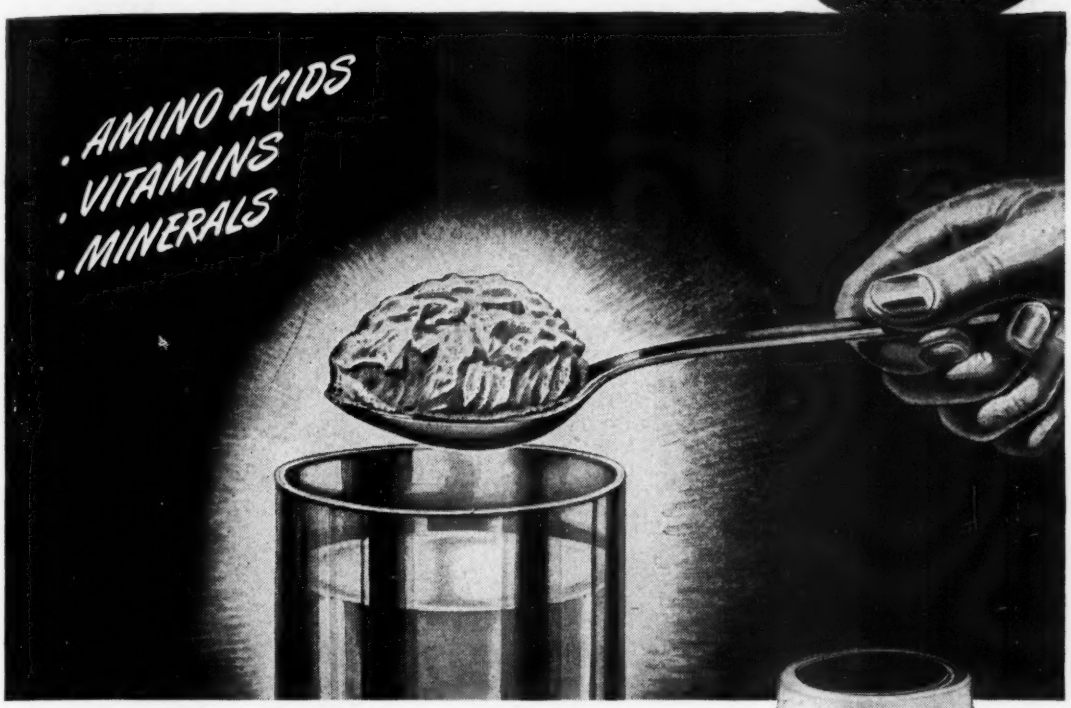

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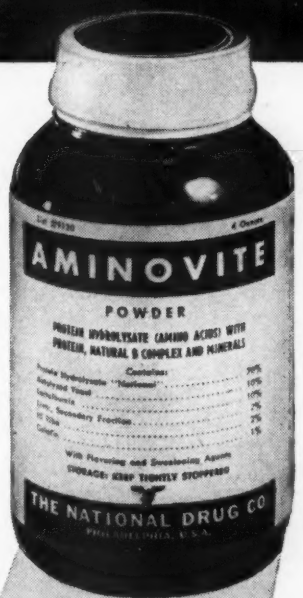

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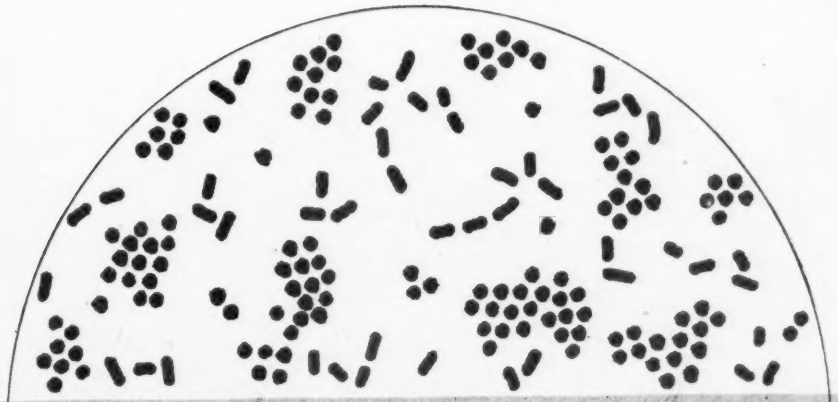

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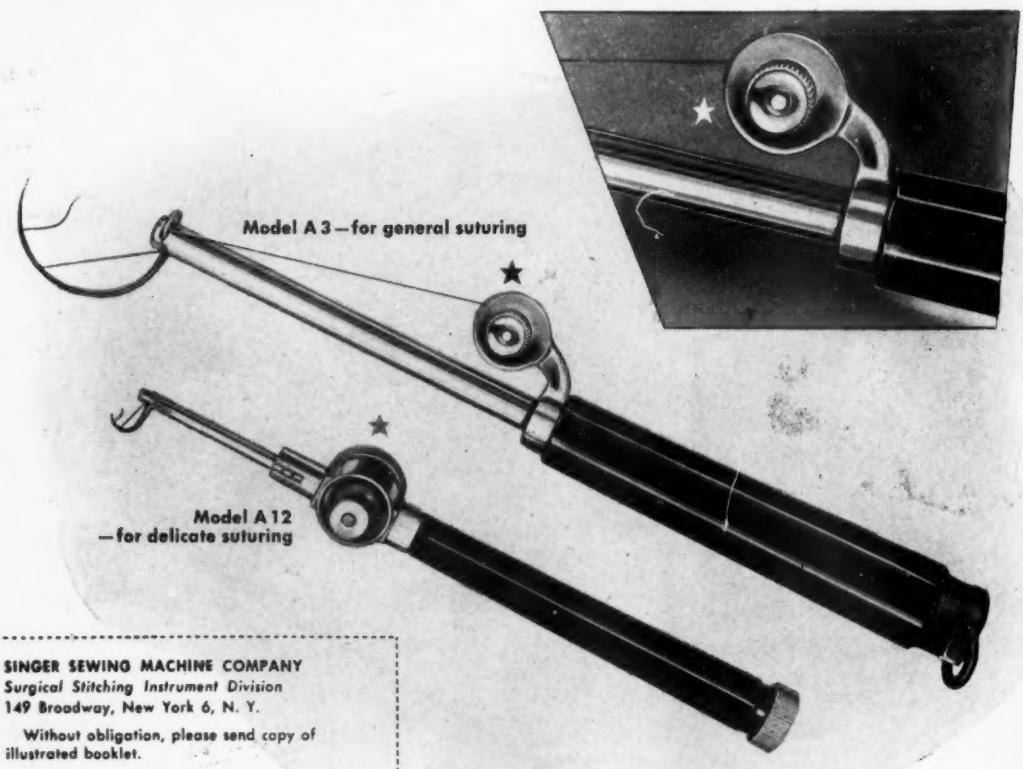

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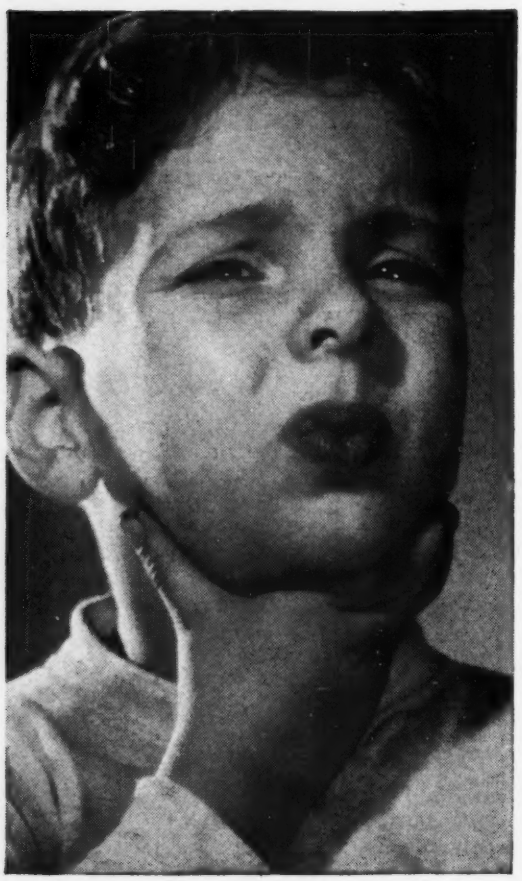

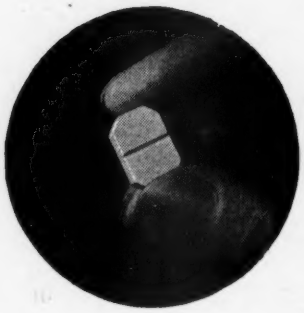

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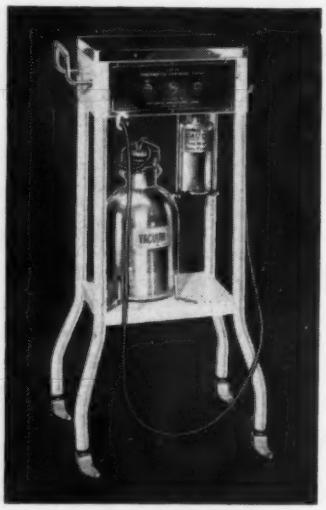

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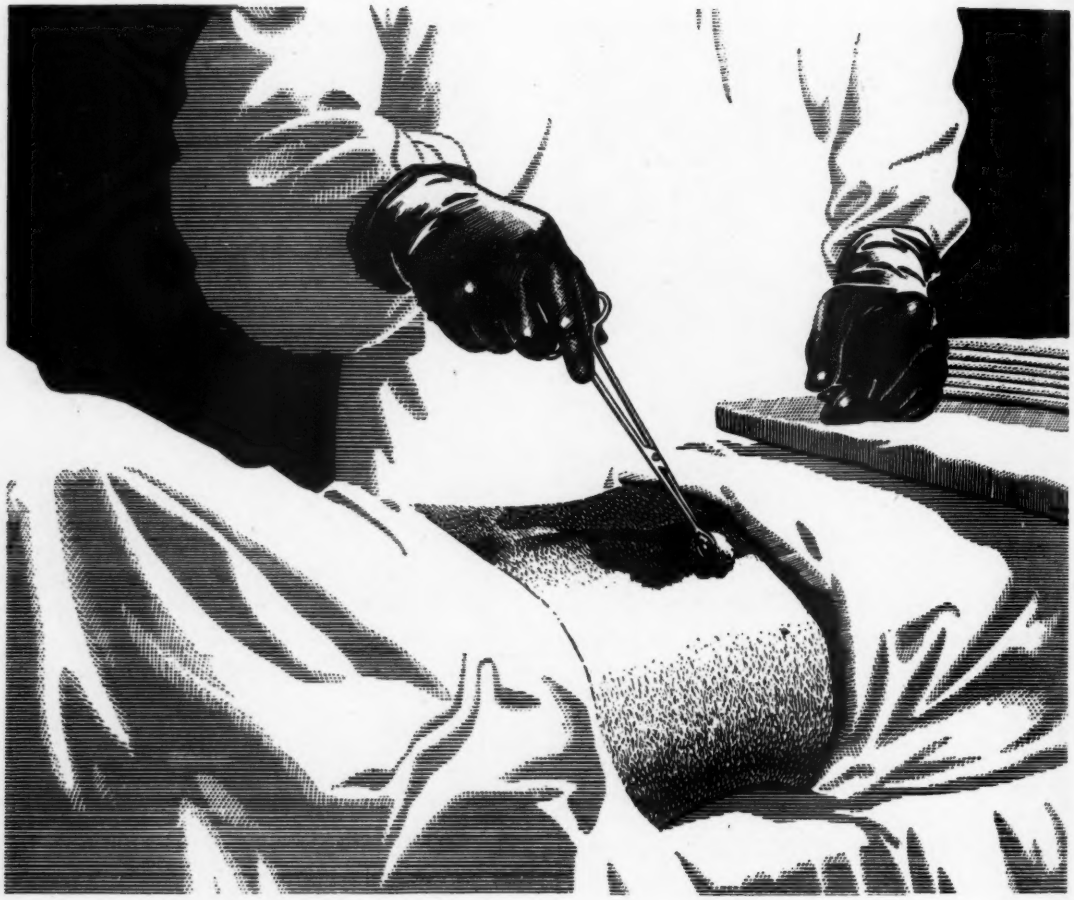

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Walter Edward Dandy
(1886-1946)

SURGERY

VOL. 19 MAY, 1946 NO. 5

Dr. Walter E. Dandy Birthday Number

WALTER EDWARD DANDY

1886-1946

WALTER EDWARD DANDY died on Friday morning, April 19, 1946. The original suggestion that this issue of SURGERY be dedicated to him on the occasion of his sixtieth birthday came from friends in Australia. This suggestion met with the hearty approval of the editors of SURGERY. It was hoped that this series of papers would be published in April since Dr. Dandy's birthday was on the sixth of this month but several factors led to delay. In the meantime the untimely death of this great man has shocked the medical profession and thousands of his patients and friends.

Walter Edward Dandy, the son of John and Rachel Dandy, was born in Sedalia, Mo. He received a Bachelor of Arts degree at the University of Missouri in 1907 and was graduated from the Johns Hopkins School of Medicine in 1910. He served as a house officer in surgery at the Johns Hopkins Hospital during the succeeding eight years, the last two of which he was resident surgeon. In this capacity he followed Dr. Roy D. McClure and Dr. George Heuer and preceded the late Dr. Mont Reid.

The busy years as a house officer sharpened rather than dulled Dr. Dandy's interest in investigative work. Dr. Halsted recognized his unusual gifts and encouraged and stimulated him. In 1913, three years after his graduation, Dr. Dandy reported his fundamental studies on hydrocephalus, which added much to the knowledge of the pathogenesis and the treatment of this disorder. Dr. Halsted remarked to Dr. E. A. Park at the time, "Dandy will never do anything equal to this again. Few men make more than one great contribution to medicine." Subsequent events showed that Dr. Halsted had underrated the ability of his brilliant pupil.

In 1918, at the age of 32 years, Dr. Dandy made his greatest contribution. While viewing the x-ray film of the abdomen of a patient, he observed the difference in the appearance of shadows which were cast by air and by fluid. It occurred to him that the outline of the ventricles of the brain could be visualized if some of the fluid were removed and replaced by air. The pro-

cedure was used first on animals and later on patients. It was found that an abnormality in contour of one of the ventricles of the brain as seen on the x-ray film frequently revealed the location of lesions which could not be determined by other means. At first this method was not received with enthusiasm by most neurosurgeons, but it is used now in all medical centers and is regarded by many as the greatest advance ever made in neurosurgery.

Dr. Dandy's original work did not cease with this second contribution. In 1925 he described an operation for trigeminal neuralgia which reduced the likelihood of postoperative corneal anesthesia and ulcers. Shortly thereafter he developed a similar operative procedure for the treatment of glossopharyngeal neuralgia. In 1928 Dr. Dandy devised an operation for the treatment of Ménière's disease, which is regarded by many as his second most important contribution. He discovered that the attacks of vertigo could be eliminated by division of fibers of the anterior part of the acoustic nerve and that hearing was not impaired. No satisfactory treatment for this condition existed prior to this discovery.

In 1928 Dr. Dandy described the findings, together with the operative treatment, in several patients with ruptured intervertebral discs. He did not, however, appreciate the surgical significance of this disorder until the important work of Barr and Mixter was published. Following this he took a renewed interest in the subject and demonstrated the frequency with which a ruptured disc causes pain in the lower back and leg. New diagnostic tests were devised and operative therapy was improved.

Other achievements of Dr. Dandy's include the operative treatment of intracranial aneurysms, a field in which he alone achieved much success for a number of years. His operations for tumors of the acoustic nerves and for large lesions of the frontal lobes are almost equally outstanding. His book on the treatment of orbital tumors is a classic. At the time of the onset of his recent illness he was engaged in experimental and clinical studies on the so-called "conscious" center. It may be said truly that his interest and enthusiasm never waned and his imagination and genius led him from one discovery to another. Dr. Halsted once said to Dr. Abraham Flexner and to others that he regarded Dr. Dandy as the most brilliant pupil he ever had.

Despite his busy professional life, Dr. Dandy found time to play golf and tennis and his interest in sports in general was keen. He took great pride in a helmet which he devised for protecting the heads of baseball players. He was fond of bridge and was a good though somewhat unorthodox player. Dr. Dandy read extensively and was an authority on the history of the Civil War period.

An unusually happy home life was Dr. Dandy's greatest source of pleasure. The congenial life there meant more to him than any outside attractions, and it was very seldom that he could be induced to leave his home and family in the evening. He kept constantly in touch with the interests of his wife and children. Walter Jr. is a third year student at the Johns Hopkins Medical School, Mary Ellen a freshman at Wellesley, and Kitty Lou and Margaret

attend private schools in Baltimore. The interesting conversations and the pleasant give and take between parents and children were delightful.

Many knew of Dr. Dandy's eminence as a neurosurgeon; few were acquainted with his generosity and his deep interest in students and associates. The cold exterior which a neurosurgeon of the formative days of this specialty had to assume if he were to continue in the discouraging work was only superficial. The student in need was aided in paying his tuition, the sick house officer or nurse was sent to a resort for a vacation, the orderly was given a home, the surgical department received an anonymous gift. Only the recipients knew of these acts of kindness and generosity.

Dr. Dandy possessed a fortunate combination of qualities: character, a clear-thinking brain, industry, an intuitive imagination, independence of thought and action, fearlessness and daring, manual dexterity, and a colorful personality. In a recent editorial in the *Baltimore Evening Sun* it was said, "He had imaginative genius to conceive of new and startling operative techniques, courage to try them, and skill—superb skill—to make them successful." It can be said without fear of contradiction that Medicine has lost one of its greatest men.

—*Alfred Blalock*

AN APPRECIATION

WALTER DANDY'S achievements in the field of neurosurgery are epitomized in this issue of SURGERY by his associates and former pupils on his sixtieth birthday. It would be of the utmost value both to neurosurgeons and to those whose plan it is to enter this field were Doctor Dandy's articles, monographs, and books to be compiled into a complete set of volumes. These contributions are truly monumental.

As one of Professor Halsted's residents Doctor Dandy was well trained as a general surgeon, and was introduced early into the surgical research laboratory. This background has contributed greatly to his eminence as an investigator, teacher, and neurosurgeon.

One of his earliest and most unique and significant contributions to neurosurgery is the procedure of ventriculography and encephalography. It was conceived when he, as resident surgeon, noted the presence of air beneath the diaphragm in a roentgenogram of a typhoid patient with perforation. The contrast medium of this air suggested to him its use in replacing cerebrospinal fluid to outline the ventricles and subarachnoid spaces.

His early and sustained interest in hydrocephalus is again shown in a recent article. He has advanced the technique of approach to lesions of the cranial nerves, acoustic neuromas, and third ventricle and other tumors. These are detailed in his comprehensive chapter on the brain found in *Lewis' Practice of Surgery*.

I am primarily interested in calling attention to Doctor Dandy's prestige as a teacher. In exacting integrity, alert cerebration, and paramount interest in the welfare of each patient, he gave in return a share in the diagnostic, operative, and postoperative care of the patient, dependent only upon the ability of his assistant. Not content with existing procedures when these seemed to be inadequate, his keen observation and deduction often led to a solution which meant the saving of a life in an apparently hopeless situation.

His outstanding success in the accurate localization of brain lesions was in each case due to a most careful consideration of the clinical and neurologic findings, with comparative study of the ventriculograms, which had in some cases been repeated once or even twice.

Sustained investigative interest in the research laboratory, complete honesty in the interpretation of experimental findings, precise clinical diagnosis, and analysis of pre- and postoperative procedures—these precepts imply certain responsibilities for the training of others, and appreciation that can never be fully expressed.

—Frederick Leet Reichert
San Francisco, Calif.

EVOLUTION OF NEUROSURGERY THROUGH WALTER E. DANDY'S WORK

DAVID FAIRMAN, M.D., BUENOS AIRES, ARGENTINA

"WHEN I was Dr. Cushing's resident, during the years 1911 and 1912, two intracranial tumors were removed, one survived." This fact, told by Walter Dandy to his pupils in a lecture delivered this year, gives an idea of the situation of neurosurgery of that time. At that period in the development of the surgery the localization of brain tumors was largely guesswork, and most tumors were found by trial and error, with only occasional successes. The usual procedures were explorations and decompressions to relieve intracranial pressure. It was hoped that eventually localization would appear before the patient's death.

Dr. Dandy's discovery of ventriculography made it possible to diagnose accurately nearly all tumors and, when present, to localize them with precision. Exploratory cranial operations were no longer justified, decompressions were eliminated, and exposures of brain tumors were made with accuracy because their location was known beforehand.

From 1910 to 1917, among Dandy's different works we have the series of brilliant and thorough experimental, pathologic, and clinical studies of hydrocephalus. Before his discovery of the cause of hydrocephalus, it was described in all textbooks as an idiopathic disease, and therefore no reasonable therapy was used. Dandy's experiments on animals by placing an obstruction in the aqueduct of Sylvius, foramen of Monro, or cisterna, reproduced every type of hydrocephalus. He discovered the place and manner of formation and absorption of cerebrospinal fluid. He finally checked the experimental studies by pathologic studies on human material. As a result, hydrocephalus has been emancipated from the diminishing group of so-called idiopathic diseases. Dandy's fundamental statements were as follows:

1. Fluid forms in the cerebral ventricles from the choroid plexuses but is not absorbed there.

2. Fluid is absorbed from the entire subarachnoid space over the cerebral and cerebellar hemispheres and spinal cord; the major part of the absorption is over the cerebral hemispheres. Absorption is directly and solely into the blood by way of the capillaries of the pia arachnoid, it is not through the pacchianian granulations or stomata as taught in most textbooks of anatomy and physiology.

3. There must, therefore, be free escape of fluids from the ventricles into the cisterna magna and into the subarachnoid spaces over the cerebral hemispheres. If an obstruction exists at any point between the place of formation and place of absorption of fluids, the cerebrospinal fluid will continue to accumulate up to the point of the obstruction, hydrocephalus therefore resulting.

4. Clinical test: In order to know whether the obstruction is in the ventricles or subarachnoid space, an inert colored solution such as phenolsulfonphthalein is injected into the spinal canal and later tested for in the fluid of the lateral ventricles. If the dye does not come through, the block is in the ventricular system; if it does come through, the block in is the cisternae.

5. The three operations which were devised were based on this accurate anatomic, physiologic, and clinical knowledge: (a) Third ventriculostomy (floor of ventricle) to be used for obstruction at the aqueduct of Sylvius. (b) Reconstruction of new foramen at fourth ventricle when the foramina of Luschka and Magendie are occluded. (c) The bilateral removal of the choroid plexuses from the lateral ventricles when hydrocephalus is due to a cisternal block, and later, if necessary, cauterization of choroid plexuses in foramina of Luschka and Magendie.

Diagnosis and Treatment of Strictures at the Aqueduct of Sylvius (the Iter).—In July, 1945, Dandy published his work "Diagnosis and Treatment of Strictures of the Aqueduct of Sylvius (Causing Hydrocephalus)." In this work he gave the results of his experiences of nearly twenty-five years in this group of hydrocephalics.

In 1922, Dandy proposed an anterior third ventriculostomy in the floor of the ventricle through a small supraorbital approach.

In 1932, he substituted a lateral approach to the third ventricle by the temporal route and this has since been used exclusively.

Dandy demonstrated that structures of the aqueduct of Sylvius are diagnosed in infants and children by (1) the dye test indicating an obstruction, plus (2) a low inion, and (3) usually without ventriculography.

In his recent work Dandy made the following fundamental statements: (1) An oversized head with Macewen's sign (cracked pot sound) and (2) an inion normally placed or at times lower than normal indicates that the hydrocephalus is due to a stricture of the iter. (3) If the inion is higher than normal under the same conditions, the obstruction is at the foramina of Luschka and Magendie (because the enlarged fourth ventricle, or tumor, pushes the inion upward).

Third ventriculostomy, or temporal approach, is the operative choice for hydrocephalus due to a stricture of the aqueduct. It is much superior to the anterior approach because (1) external hydrocephalus does not follow; (2) it is not necessary to section the optic nerve; (3) the scar is under the hairline.

Cures are attained in most cases when the patient is more than 1 year old and in less than one-half the cases of infants.

Excellent results were obtained in twenty-nine cases (patients over 1 year of age). From this group twenty-four are still living and cured. The time since operation is between six months and one year in two cases; between five and eleven years in eleven cases; and, finally, between ten and twenty-three years in four cases.

The Treatment of Obstructions of the Foramina of Magendie and Luschka.—In 1921, Dandy published "The Diagnosis and Treatment of Hydrocephalus Due

to Occlusions of the Foramina of Magendie and Luschka." In this work, Dandy described a group of cases of hydrocephalus caused by closure of the foramina of Luschka and Magendie, showing the pathology by post-mortem specimens, describing the means by which it can be diagnosed clinically, and introducing his operative procedure to produce its cure.

Dandy stated that there are two types of occlusions of the foramina of Luschka and Magendie: One, which occurs congenitally, may or may not be the result of an intrauterine inflammation; the second, which occurs at all ages, always follows some form of meningitis.

Dandy demonstrated from post-mortem examinations that the closure of the foramina of Luschka and Magendie in the same brain will, without exception, result in hydrocephalus because there are no collateral channels by which the fluid can be carried from the ventricles to the subarachnoid spaces.

Ventriculography.—Although each of Dandy's work establishes a landmark in the history of neurosurgery, his discovery of the ventriculography in 1918 started a new epoch, not only from the technical point of view but from the intellectual, since it transformed radically and integrally the mental conception of neurosurgery, replacing the existing dogmatism and empiricism with a realistic judgment and authentic scientific criterion.

It was in the same year, 1918, that the most prominent Argentine surgeon, Dr. Ricardo Finochietto, visited Dr. Dandy, seeing him perform in one day five operations, using in each a technique that he had devised. On that date began the cordial friendship between the two masters, to be so profitable later for the progress of Argentine neurosurgery, through their pupils. Through this friendship I became one of Dr. Dandy's pupils.

In 1920 Dandy published his work, "Localization or Elimination of Cerebral Tumors by Ventriculography." In this he made the following statements:

"Ventriculography is invaluable in the localization of obscure brain tumors. So-called unlocalizable tumors comprise at present over half of the total number."

"Practically all brain tumors either directly or indirectly affect some part of the ventricular system."

"Local changes in the size, shape, and position of one or both ventricles as shown by the ventriculogram will accurately localize most obscure tumors of either cerebral hemisphere."

"Every effort should be made to localize the tumors before resorting to any operative procedures."

"The usual subtemporal decompression is useless and dangerous when a hydrocephalus is present, i.e., when the tumor is in the brain stem or cerebellum."

"A suboccipital decompression (cerebellar operation) is extremely dangerous when the lesion is in the cerebral hemisphere."

"To differentiate between cerebral and cerebellar lesions is frequently one of the most difficult tasks in intracranial localization. Ventriculography at once separates these two groups and indicates the operation of choice."

In later works Dr. Dandy propounded the following fundamentals of modern surgery of the brain:

"Brain tumors are among the most frequent neoplastic lesions in the body; their growth is always progressive and almost always leads to a train of terrible sequelae and eventually to death."

"There is only one form of treatment of tumors of the brain, operative removal, and this must be complete."

"To obtain the best operative results, brain tumors must be diagnosed and localized in the earliest stages."

"It is now possible to diagnose and localize practically every tumor and in the early stages. When all other signs and symptoms fail in the localization, cerebral pneumography will make the diagnosis and localization with precision and without equivocation; and when a tumor is not present, it can be excluded by the same method."

"The operative approach will be dictated by the precise localization. The approach should afford adequate room, and it should be directly over the tumor. After correct localization all brain tumors should be disclosed at operation."

"Every effort should be made to cure the patient by complete extirpation of the growth. There is less mortality from carefully performed tumor extirpations than from unsuccessful explorations for tumors. When, for any reason, it is impossible or unjustifiable to remove the tumor, the maximum palliation should be given at the same operation."

"Decompressions routinely performed are among the most harmful and indefensible operations in surgery. They should never be performed for unlocalizable tumors; they are the exact equivalent of giving morphine for abdominal pain. The symptoms are masked until it is too late. Decompression should be performed only as a last resort when the tumor cannot be removed and then only after the location of the tumor is known; for in half the cases of brain tumor, no good can possibly be derived from a decompression."

"About 50 per cent of brain tumors can be localized by a neurological examination, and about 15 per cent more can be localized with the x-ray without air injection. In the remaining 35 per cent the injection of air can alone make the localization. It is a dangerous proceure and should be used only when necessary."

Today, twenty-five years later, these revolutionary statements form the background of modern neurologic surgery.

Acoustic Tumors.—In 1922 Dandy published a preliminary report and in 1925 a complete work, entitled "An Operation for the Total Removal of Cerebello-Pontine (Acoustic) Tumors." Considering that most frequent tumor of the cerebello-pontine angle is the acoustic neuroma, which is a potentially benign lesion usually easy of recognition, nevertheless it had presented surgical problems which were well nigh insuperable. At the International Congress of Medicine in London in 1913, the three great European surgeons, Horsley of London, Eiselsberg of Vienna, and Krause of Berlin, who had in such large measure been responsible for the birth and growth of brain surgery, presented their results on the extirpation of cerebellopontine tumors to that date. Horsley had ten opera-

tive deaths in fifteen cases (67 per cent), Eiselsberg had thirteen deaths in seventeen cases (77 per cent), and Krause had twenty-six deaths in thirty-one cases (84 per cent). Krause admitted that they yielded the poorest results of all the brain tumor operations. All of the accumulated technical advances of a quarter of a century had made no improvement in the results. At any rate the continuance of an operation carrying such an astounding mortality after such an exhaustive trial was impossible. The reaction came with the publication in 1917 of Cushing's important monograph on acoustic tumors, and with it a modified treatment. He accepted the only conclusion which the foregoing results and experience of his own could justify, that is, "I doubt very much, unless some more perfected method is devised, whether one of these tumors can ever with safety be totally enucleated." He no longer attempted to enucleate these tumors totally but was content to offer a method by which the tumor could be removed partially (intracapsular enucleation). Cushing's contribution was the only important advance in the treatment of cerebellopontine tumors up to that time.

In 1925 Dandy presented his operative procedure by which cerebellopontine tumors can be removed completely. After a thorough and carefully guarded intracapsular enucleation, the capsule of the tumor is painstakingly dissected from the brain stem. In 1934 Dandy introduced the unilateral approach greatly simplifying the procedure. About this procedure Horrax said: "The unilateral approach and total removal has become accepted more and more as the best mode of attack. This method was described first by Dandy in 1922 and enlarged upon by him in 1934."

Not only did Dandy's method allow the complete extirpation of the tumor, but also he gave a scientific explanation of why it carried the minimal mortality. He demonstrated that without doubt the danger of cerebellar decompression is proportionate to the degree of hydrocephalus which is present at the time of operation, and that the removal of the occipital bone at once liberates the pressure in the posterior cranial fossa. But this benefit is at once countered, and may be greatly exceeded, by the injurious effects of the backward pressure on the tentorium (hydrocephalus) and its full force is now exerted without opposition upon the delicate brain stem, jamming it backward. He also demonstrated that when the tumor is removed, the obstruction of the aqueduct of Sylvius is released, and the hydrocephalus automatically cured and its ill effects obviated. This obtains only at times with subtotal intracapsular enucleation.

In his publication of 1934, Dandy made the operation much simpler and safer by removing the outer cap of the cerebellum before beginning the extirpation of the tumor.

And at last he demonstrated that the operation which at once removes the cause not only carries the lowest mortality, but at the same time offers the best immediate and only permanent results because these tumors do not recur. The mortality in removing these tumors is now less than 3 per cent.

Tumors in the Third Ventricle.—Up to 1921 a most interesting and important group of benign encapsulated tumors lying within the third ventricle had remained outside the fields of diagnosis and treatment, and therefore the patients with this kind of tumor were condemned to die. On Sept. 30, 1921, the

discoverer of the ventriculography was the first to demonstrate that with the mechanical evidence of the ventriculography it was possible to diagnose and localize the tumors of the third ventricle with the greatest accuracy and certainty, a fundamental prerequisite for their surgical attack.

On October 18 of the same year Dandy was the first to perform an operation extirpating completely a tumor of the third ventricle, with an excellent result.

Later from his surgical experience he collected twenty-one cases, publishing in 1933 his book, *Benign Tumors in the Third Ventricle of the Brain: Diagnosis and Treatment;* all of these tumors had been disclosed at operation and removed.

Up to 1933, from the literature forty-seven tumors of similar character had been reported, but all of these were post-mortem findings, none having been correctly diagnosed during life.

In his book, Dandy presented the three methods of operation that he devised: (1) by the pineal route (posterior approach), (2) by the hypophyseal route (anterior approach) with or without resection of the frontal lobe, and (3) along the midline and anterior to the Rolandic area, used for cysts of the cavum septi pellucidi and cavum Vergae.

Tumors in the Lateral Ventricles.—The first primary benign tumor in the lateral ventricle to be found at operation and completely removed was reported by Dandy in 1920. The patient is still living.

The localization was made by ventriculography (on Oct. 23, 1918), after three attempts to find the tumor had been unsuccessful. This happened to be the first brain tumor to be localized by this method.

In 1934 Dandy published his book, *Benign, Encapsulated Tumors in the Lateral Ventricle of the Brain: Diagnosis and Treatment.* In this book thirteen benign encapsulated tumors in the lateral ventricles of the brain are reported, all of which had been disclosed at operation and removed. None of the twenty-five additional cases found in literature had been diagnosed during the life of the patient or removed at operation. He demonstrated that the encapsulated tumors in the lateral and third ventricle are most rewarding exceptions to the ill-founded general rule, which long held neurologic surgeons in restraint, that "every tumor below the surface of the brain is a glioma." Dandy stated that the wisdom of such a rule in the days before accurate localizations of intracranial tumors cannot be questioned, but at the present time a far better dictum is "actually to see every brain tumor (there are occasional exceptions) and to know whether it can or cannot be removed." There are now no tumors giving signs or symptoms that cannot be diagnosed accurately, localized precisely, and disclosed at operation. He demonstrated also that none of the tumors in the lateral ventricles of the brain can be localized from the analysis of the signs and symptoms but all can be localized with absolute precision by means of ventriculography.

Operations for Trigeminal Neuralgia, Ménière's Disease, and Glossopharyngeal Neuralgia.—

Trigeminal Neuralgia: While developing his operation for the removal of acoustic tumors, Dandy saw the possibility of using the same cerebellar route for

the treatment of trigeminal neuralgia. In February, 1925, he presented his operative procedure by which the sensory route of the trigeminal nerve was sectioned alongside the pons and discovered that all the liabilities of the old operative procedure were avoided. In 1932, Dandy pointed out the following advantages, based on 250 cases, of the subcerebellar route:

1. In no instance had there been postoperative keratitis. The reason for the absence of keratitis, which is one of the principal complications of the temporal route, is that only an instant is required to divide the nerve after exposure under the cerebellum. The keratitis was trophic, as a result of operative trauma to the gasserian ganglion, and this sequel was not due to loss of sensation as had been supposed.

2. The motor root of the nerve was never injured because it is at a greater distance from the sensory root than at any other point of its course. This is all important in cases of bilateral tic douloureux.

3. The seventh nerve was not injured, because it was a safe distance from the fifth nerve.

4. In one out of every twenty cases (5 per cent) a tumor was the cause of the neuralgia and would have been missed if the temporal route had been used. When the tic douloureux is caused by a tumor, it is impossible to differentiate by clinical tests.

Analyzing brain tumors, affecting the fifth nerve, Dandy demonstrated that always the tumor is on the sensory root of the fifth nerve—there being many of them, he found that there is never one on the peripheral branch of the nerve that causes this pain; never is there one at the gasserian ganglion when the attacks are typical.

Dandy made the fundamental statement that only a lesion in the upper neuron, that is between the ganglia of the nerve and the brain stem, can give a paroxysmal pain. In view of this fact, unnecessary peripheral treatments are avoided and the frequently assumed causal relationship of trigeminal neuralgia to peripheral infections is eliminated.

Trigeminal neuralgia has for many years been regarded as idiopathic, but in 1940 Dandy, on the basis of over 500 cases, demonstrated that nearly always there is a very definite underlying cause for it. In 5 per cent of the cases, a tumor or anuerysm will be in the cerebellopontine angle, pressing upon the fifth nerve and causing this pain. In nearly all of the remaining cases an artery will be found on either the under surface or the outer surface of the sensory root.

It is the pressure of this artery upon the bare sensory root that causes the tic pain. This explains why the pain appears in the later part of life; it is when the artery hardens from sclerosis.

Furthermore, in later works, Dandy gave the explanation of a fundamental physiologic fact. When the posterior one-half or even three-fourths of the sensory root of the fifth nerve is sectioned, it is possible to preserve practically the entire sensation of the face and at the same time to cure the pain, regardless of the branch or branches involved. This is due to the fact that, with few exceptions, all the pain fibers are collected in the posterior half of the sensory tract.

Sensations to the face are preserved when three-fourths of a nerve is divided, because of the redundancy of fibers carrying the same function.

Ménière's Disease: In 1928, Dandy devised an operation for the surgical treatment of Ménière's disease, publishing a preliminary report with nine cases.

By 1945 Dandy had operated upon the auditory nerve 682 times for Ménière disease, with one death due to meningitis.

At first the nerve was totally divided, but in 1933 Dandy demonstrated that the vestibular branch of the nerve can be divided leaving the auditory branch and consequently not impairing the hearing, in fact, Dandy found that division of even three-fourth of the auditory division of the nerve does not affect the hearing, apparently because of redundant fibers carrying similar functions, and this permits a good margin of safety in insuring destruction of all vestibular fibers that may be included with the auditory fibers.

Dandy stated that in about 10 per cent of the patients with Ménière's attacks both nerves are affected and he demonstrated these facts:

1. After both vestibular nerves are sectioned the gait is normal and the Romberg negative.

2. There are two aftereffects: (a) Jumbling of objects when the patient is in motion; when at rest objects are stationary. (b) Uncertainty, when the patient is walking in the dark.

3. These two disturbances are due to the very intimate association between the vestibular and the visual apparatus in human beings.

At the present time most textbooks carry the statement that Ménière's disease is due to a hemorrhage in the internal ear (Ménière's original demonstration in one case) but Dandy demonstrated the following fundamental facts:

1. Since both the vestibular and cochlear divisions of this nerve are involved, it is evident that the lesion is in the nerve itself and cannot therefore occur in the semicircular canals.

2. The causative lesion must be in the nerve and not in the end organs because the attacks are paroxysmal.

3. The attacks of Ménière's disease are unquestionably similar to the paroxysmal attacks in trigeminal neuralgia, glossopharyngeal neuralgia, and epilepsy.

4. A peripheral lesion cannot cause recurring paroxysmal attacks. Always a higher neurone, and never a peripheral nerve or end organ, is the site of the lesion causing paroxysmal attacks.

5. The cases of vestibulitis following mastoid infection are quite dissimilar in that the dizziness is continuous until the infection is over, and then does not recur in paroxysmal attacks.

The pathology of Ménière's disease has been studied rarely and the few reports have been concentrated upon the semicircular canals. In 560 cases exposed at operation, Dandy found (1) tumors at the vestibular nerve in ten cases (2 per cent), (2) an encircling artery constricting the nerve or a large artery lying upon it in 20 per cent, and (3) no gross lesion visible in the remaining cases.

Dandy called it pseudo-Ménière's disease when they are exactly the same dizzy attacks, but without unilateral loss of hearing and without tinnitus. Dandy

stated that it is quite probable that pseudo-Ménière's disease is, at times at least, only a beginning Ménière's disease in which the tinnitus and deafness have not yet appeared.

In 1939, Dandy published an experimental study of the central connections of the vestibular pathways. He found that the results following section of the vestibular nerves in animals are completely different from those obtained in human beings, therefore, their transfer to man is impossible. In this experimental work Dandy demonstrated five points:

1. Section of either eighth nerve results in frequent spells of violent whirling of the body always to the side of the lesion.

2. Section of both nerves results in violent whirling to either side.

3. After removal of half or all of the cerebellum there is no whirling movement.

4. After section of the eighth nerve and removal of the cerebellar lobe of the same side, violent rotation follows and the whirling is to the same side.

5. After section of one eighth nerve and removal of the opposite cerebellar hemisphere no whirling results. The central control of whirling movements, therefore, is contralateral to the eighth nerve.

Glossopharyngeal Neuralgia.—Up to 1927, the operations employed in glossopharyngeal neuralgia had been of the peripheral type. Besides the difficulties of isolating the ninth nerve in the neck without injuring the vagus, the objection remained that regeneration of the nerve is certain to follow its division peripherally.

On April 6, 1927, Dandy was the first to perform an intracranial section of the ninth nerve in the posterior cranial fossa for this disease—an operation almost devoid of danger, and productive of permanent cure without disability.

The first accurate knowledge of the function of the glossopharyngeal nerve was obtained by Dandy when he divided the nerve intracranially.

Isolated loss of function of the ninth nerve had not been known. All previous observations had been made on relatively gross injuries either from tumors or trauma. In all of these some fibers of the vagus had been injured as well as the glossopharyngeal nerve, and functions had erroneously been accredited to the latter, which in reality belonged to the former.

Dandy demonstrated that there was no perceptible motor function in the ninth nerve, the pharyngeal muscles being normal after its intracranial section.

Anatomists state that the stylopharyngeus muscle is the only muscle supplied by the ninth nerve but Dandy showed that paralysis of this muscle cannot be demonstrated by any loss of function.

Dandy stated that the ninth nerve supplies taste for the posterior third of the tongue, but does not supply taste to the whole tongue, a view long held by Luciani and by many physiologists even at present.

The sensory distribution of the glossopharyngeal nerve was shown by Dandy to be in the posterior third of the tongue, the anterior lateral and posterior walls of the pharynx, the vallecula, and the pyriform sinus.

Cure of Pain in Malignant Lesions of the Mouth.—In July, 1929, Dandy published his work "Operative Relief From Pain in Lesions of the Mouth,

Tongue and Throat.'' In this Dandy introduced an operation for the complete and permanent relief from pain associated with chronic and malignant lesions of the mouth, nose, tongue, and throat. He stated that there is no other way of permanent relief, when the pain is referable to the sensory domain of both the glossopharyngeal and trigeminal nerves on one side.

Dandy demonstrated that (1) under such conditions the glossopharyngeal nerve and the sensory root of the trigeminus can be divided at the brain stem (Dandy's subcerebellar approach), both nerves being exposed simultaneously in the same operative field; and (2) both nerves can be divided easily, quickly, and with practically no danger to life of function.

Operation for Torticollis.—In April, 1928, Dandy devised an operation by which the first, second, and third cervical motor roots were divided intraspinally on each side and both spinal accessory nerves were sectioned in the neck, all at the same operation. He published this work in 1930 under the title ''An Operation for the Treatment of Spasmodic Torticollis.''

The only modification that Dandy since added to the procedure is to include the fourth cervical motor nerve on both sides. He demonstrated that this definitely adds to the percentage of cures and does not affect the phrenic nerves.

Recently Dandy pointed out the excellent results obtained in a series of fifty operations that he performed.

Dandy stated that in most instances spasmodic torticollis is undoubtedly of organic and not of functional origin.

Epilepsy.—Epilepsy had always been considered as an idiopathic disease.

Dandy's experiments on animals, clinical impressions on human patients, observations from a series of operations performed by him on the brain of patients with epilepsy, and post-mortem studies of their brains, have entirely emancipated epilepsy from the diminishing group of so-called idiopathic diseases.

In 1925, through a series of experiments on animals Dandy demonstrated these points:

1. That when the motor cortex of dogs or cats had been injured by an incision or by inclusion of a foreign body, spontaneous convulsions appeared from time to time thereafter.

2. That subsequently after a period of many weeks and months convulsions could be induced by absinth, with from one-third to one-seventh of the dose required to produce convulsions in the normal animal.

3. That injuries to other parts of the brain did not produce results so striking, but suggested a lowering of the convulsive threshold.

4. That the motor cortex once injured thereafter lowers the threshold at which convulsions appear.

5. That in these animals, therefore, just as in human beings, the margin of safety which is the difference between the normal and the epileptic condition is permanently lost when a cerebral lesion of long standing exists.

From a close study of a large series of patients with epilepsy resulting from tumors of the brain, Dandy observed that clonic convulsions, whether Jacksonian or generalized, are due to those tumors which are localized in the cerebral hemispheres, that is, excluding the brain stem and cerebellum.

The series of experiments on dogs, reported by Dandy in 1927, explains the reasons for these conclusions. In these experiments Dandy demonstrated several facts:

1. Clonic convulsions were induced by faradic stimulation of the motor cortex or subcortex but not cerebellar hemispheres.

2. When, during a localized clonic convulsion, the motor cortex of the controlateral side is excised by a sweep of the knife, the convulsion instantly and completely ceases.

3. When, during a generalized clonic convulsion, the motor cortex of one side is excised by a sweep of the knife, the contralateral side instantly ceases to convulse, but the course of the convulsion on the opposite side is unaffected.

4. When, during a generalized convulsion, the motor cortex of both hemispheres is excised by two rapid sweeps of the knife, the convulsion instantly ceases in all parts of the body.

5. When, after excision of the motor cortex of both cerebral hemispheres, the subcortical motor tracts of either side are stimulated with stimuli of any intensity, clonic convulsions either localized or general could not again be induced in any part of the body.

6. After excision of the cerebellum, stimulation of the intact motor cortex produces clonic convulsions precisely as before the cerebellum was removed.

By these experiments, Dandy showed that (a) only stimulation of the motor cortex or its subjacent fibers (with the motor cortex intact) can produce clonic convulsions; (b) this specificity of function similarly obtains in the human motor cortex and is probably a function of the pyramidal cells; (c) clinical evidence indicates that the attacks of epilepsy, of which the convulsions are a part, may or may not begin in the motor cortex but without the motor cortex, the convulsions could not develop.

In 1925 Dandy emphasized the close analogy between epilepsy and trigeminal neuralgia. Furthermore, in later works, Dandy made the following fundamental statements:

1. The attacks of epilepsy are precisely similar to the paroxysmal attacks in trigeminal or glossopharyngeal neuralgia and Ménière's disease.

2. It is impossible to produce recurring spasmodic attacks of convulsions, of dizziness, or of pain, except by a lesion in the upper neurone.

3. Involvement of a higher neurone is essential for the production of any paroxysmal attacks. Epilepsy cannot be produced by any lesion along the peripheral nerves. It can only result from a lesion in the cerebral hemisphere. From his experimental, pathologic, clinical, and surgical studies, Dandy presented a sufficient number of unquestioned facts to place epilepsy unequivocally upon a pathologic instead of an idiopathic basis.

Pineal Tumors.—Up to 1921, tumors of the pineal body had rarely been diagnosed, and almost all had been accidental findings at necropsy. The absence of a correct localization of this lesion had precluded any operative interference with this region.

In 1915, Dandy conceived an operative procedure by which it was possible to remove the pineal body in dogs. From a series of young puppies and adult dogs in which the pineal body was totally removed, Dandy observed no change in the normal growth of the body and no sexual precocity or retardation.

Based on his experimental work, Dandy devised in 1921, "An Operation for the Removal of Pineal Tumors." This was precisely the same as the experimental approach. Dandy demonstrated the following points:

1. It is possible to make a correct diagnosis and accurate localization of pineal tumors.

2. The most significant single localizing sign is bilateral ptosis.

3. Bilateral ptosis is due to downward pressure upon the quadrageminal bodies, affecting the nuclei of the oculomotor nerves in the midbrain.

4. A precise diagnosis of pineal tumors is always possible only by ventriculography, but to accomplish this end perfect visualization of the third ventricle is necessary.

5. The important differential point, by ventriculography, between a primary pineal tumor and tumors in the posterior fossa, is that pineal tumors usually cause a filling defect in the posterior part of the third ventricle.

In Dandy's operation the following methods are used:

1. The pineal tumor is exposed by an occipital approach and separation of the parietal and occipital lobes from the falx; the corpus callosum is split and the tumor dissected between the small veins of Galen, which are on each side.

2. For the exposure of tumors projecting posteriorly, it is necessary to split the tentorium, alongside the straight sinus.

3. For the very large tumors or those in which adequate room cannot be obtained by tapping the lateral ventricles the occipital lobe is resected before attacking the tumor.

Up to the present time Dandy has removed twenty pineal tumors, with a mortality of about 20 per cent.

Tumors of the Hypophysis.—Up to 1925, the removal of hypophyseal tumors by the intracranial approach had carried a very high, almost prohibitive, mortality. From his experiences with hypophysectomies in animals Dandy demonstrated several facts:

1. The greatest cause of death was injury to the brain, due to traction in an effort to overcome a limited exposure of the hypophyseal region.

2. The deaths which had been erroneously attributed to loss of the hypophysis were in reality due to cerebral trauma incident to the operation, and when a better exposure was obtained, there was almost no mortality.

3. In operations on man the traumatic cause of death was precisely the same as with animals.

Based on his accurate anatomic and physiologic knowledge, Dandy devised in 1925 the hypophyseal operation which is used today. He made four statements:

1. Avertin anesthesia is one of the most essential features of the operation, because it eliminates the swelling of the brain due to ether anesthesia.

2. The size of the bony defect is reduced about one-half and without sacrificing the room that is so important for exposing and removing the tumor.

3. Not only is the operation easier of performance but also the frequent injury to the rolandic area with resulting convulsions and hemiplegia following the larger cerebral exposure is always avoided.

4. The danger of extradural hemorrhage so common with the more extensive operation is practically eliminated.

Dandy demonstrated that the trauma to the frontal lobe can be avoided or at least greatly reduced (1) by lowering the head 80 to 90 degrees, in order to allow the brain to gravitate and reduce the force required for retraction of the frontal lobe and (2) by releasing as much cerebrospinal fluid as possible from the cisterna chiasmatis and by ventricular taps.

Dandy pointed out that the steps to be taken for actual removal depend upon several anatomic factors: (1) the length of the optic nerves, (2) the location of the tumor in relation to the optic nerves, and (3) the existence of intracranial pressure due to the tumor's bulk or due to hydrocephalus, in addition to the tumor's mass—to resolve all of these variations.

Dandy presented three procedures: (1) removal of the tumor between the intact optic nerves, (2) removal of the tumor after division of one optic nerve, and (3) removal of the tumor after resection of part of a frontal lobe and with or without division of one optic nerve.

In May, 1931, Dandy published his work "Avertin Anesthesia in Neurologic Surgery."

After a cautious beginning because of the adverse reports from Germany, where many serious results followed the pioneer efforts, Dandy demonstrated these points:

1. Not only were all the liabilities of ether entirely eliminated, but every advantage of local anesthesia was also obtained.

2. To obtain the best results with safety, an average dose should be given and any remaining deficit in the anesthesia may be overcome by supplementing ether by inhalation.

3. The amount of supplementary anesthesia, if necessary, is very small and does not change the basic character of the perfect anesthesia.

4. Owing to the entire absence of swelling of the brain, it has been possible to modify greatly the magnitude of the cranial exposure through which certain tumors of the brain are removed.

5. It is known beforehand that an extra allowance of bony defect will not be required to compensate for an increased volume of brain due to the effect of the anesthetic and therefore a smaller bone flap can be used in attacking all intracranial tumors.

In his great surgical experience, for over fourteen years, Dandy has used avertin in nearly all cranial operations of magnitude in adults, and he states that never has there been the slightest indication of an untoward result; the cranial operations are greatly facilitated by the smooth regular respirations and the absence of cerebral or cerebellar edema. This is particularly necessary in

Dandy's operations for trigeminal neuralgia and for cerebellopontine and hypophyseal tumors.

Orbital Tumors.—Up to 1920, the methods of approach to the orbit were often totally inadequate for the removal of orbital tumors. In 1921, Dandy devised an operation which permits removal of combined intraorbital and intracranial tumors. Eighty per cent of orbital tumors extend intracranially; they may arise intracranially or intraorbitally, the former more frequently. Moreover, growths that are restricted to the posterior part of the orbital cavity are much better removed by this attack.

The great advantage of Dandy's operation lies in the much fuller and safer exposure of the intraorbital contents. The optic nerve, the eyeball, three of the extraocular muscles, and the ophthalmic veins and arteries can be exposed and avoided during the dissection of the orbital tumor.

Dandy used, in his operation, the operative approach that he devised for hypophyseal tumors. He found that swelling of the brain that so commonly follows ether anesthesia is avoided when avertin is used, and for that reason the size of the bone flap necessary for the exposure of the base of the brain can be greatly reduced and simplified.

Dandy's transcranial approach carries very little risk, and it offers the maximum hope of cure without cosmetic defects.

For tumors confined to the orbit Dandy's operation offers a far better exposure of the tumor than is possible by any other method and with less injury to intraorbital muscles and nerves. Complete dissection of the tumor is much better done by this approach.

In 1941, Dandy published his book *Orbital Tumors: Results Following the Transcranial Operative Attack.* This book sets a milestone in the study of the pathology and surgical treatment of orbital tumors.

From his wide surgical experience, Dandy presented a series of twenty-four patients with intraorbital tumors operated upon by the transcranial route with a very low operative mortality (4.1 per cent). Of the series of twenty-four tumors 20 per cent were confined to the orbit and 80 per cent were combined intraorbital and intracranial growths.

Dandy stated the following facts:

1. From the clinical examination of patients with exophthalmos it is not usually possible to tell whether or not there is an intracranial extension of the orbital tumor.

2. Frequently the orbital tumor is but a small fraction of the large but silent intracranial tumor, which is usually the primary growth.

3. Only the transcranial approach will offer solution of the problem.

4. Once a tumor has been attacked by an external operative approach, the possibility of cure by a subsequent intracranial approach is greatly lessened and may be impossible.

Defective Intervertebral Disks.—In 1929 Dandy published his discovery of two ruptured lumbar intervertebral disks under the title of "Loose Cartilage From Intervertebral Disks Simulating Tumor of the Spinal Cord." These are now practically the commonest lesions on which surgery is done. These were

the first intervertebral disks disclosed at operation and recognized as such. At that time he suggested that they were probably responsible for many cases of sciatica.

In his later publications on this subject (1943-1944) he has greatly simplified the diagnosis and introduced improvements that have insured permanent cures. He and his staff have now done operations on over 2,000 disks. He disclosed the importance of the small disks which he called "concealed" disks. Dandy's concealed disks give precisely the same symptoms as the large ones, the only difference being the intensity and persistence of the symptoms, backache with or without sciatica; they comprise three-fourths of the total number. None of these can be detected by contrast media.

Dandy's method to determine which of the disks is involved includes four points:

1. The roentgenogram shows a narrow interspace in 75 per cent of the cases; narrowing of the disk is pathognomonic of a defective disk. (Until recently, defective disks were diagnosed only by injections of iodized oil, air, or other contrast mediums into the spinal cord. Dandy demonstrated that these are never necessary and are always inadvisable, frequently misleading and harmful.)

2. If there is a diminution or absence of an Achilles reflex, the disk will be at the fifth in most instances.

3. The disks at the second and third are usually localized by pain in the front of the thigh; the others give pain in the back of the leg.

4. When these tests are still negative, the affected disks can be localized at operation by horizontal pressure upon the spinous process. This determines the mobility of the joint and a defective disk will usually cause increased movement at the affected joint. The exceptions to this rule are in narrow intervertebral spaces shown roentgenologically; there may then be no mobility or less than normal.

Based on his accurate anatomic, physiologic, and clinical knowledge, Dandy devised his operative procedure proposing the thorough removal of the affected disks after which a fusion of the entire vertebral surfaces results. Dandy stated that the anatomic deviation of the lower lumbar lateral facets is the primary cause of defective disks. He demonstrated the following points:

1. His procedure does what nature attempts to do slowly over many years as is evidenced by the narrowing in the x-ray appearance.

2. After complete removal of disks the fusion of the spinal column is more nearly perfect than the most successful bone graft can be, and at the same time the cause is removed.

3. In the treatment of ruptured disks a spinal fusion by bone graft is never necessary and cannot be too strongly condemned.

In August, 1944, Dandy published his article: "Treatment of Recurring Attacks of Low Backache Without Sciatica." In this fundamental work he makes the following statements:

1. Recurring attacks of low lumbar backache without sciatica have precisely the same underlying cause as backaches plus sciatica, that is, defective intervertebral disks.

2. The diagnosis of defective disks is made solely from the history, the neurologic examination (frequently negative), and x-ray examinations of the spine.

3. Defective disks are multiple at least in 80 per cent of the cases, with or without sciatica.

4. All spinal injections for diagnosis or localization are unnecessary; they are painful and at best make the diagnosis in only 25 per cent of the cases, whereas over 98 per cent can be diagnosed and localized without them. Even simple x-ray films alone diagnose more than 75 per cent. Spinal injections of any type should be strongly condemned.

In January, 1945, Dandy published his work, "The Treatment of Spondylolisthesis." Heretofore the standard treatment for spondylolisthesis has been spinal fusions with autogenous grafts.

Dandy demonstrated these facts:

1. Subjectively and objectively, spondylolisthesis is precisely like defective intervertebral disks, only the x-ray differentiated the two conditions.

2. In at least 90 per cent, and probably all of the cases, there are two or three disks (including the one at the site of the spodylolisthesis) causing the backache and sciatica and a cure depends on the recognition and treatment of all the affected disks.

3. A cure results when the disks are completely removed. The end result of removal of the disks is fusion of the opposing vertebrae and therefore stabilization of the spine.

4. Spinal fusions by grafts are never indicated either for spondylolisthesis or for defective intervertebral disks.

Rhinorrhea and Otorrhea.—The first successful treatment of rhinorrhea was reported by Dandy in 1926. Autogenous grafts of fascia lata were sutured over the dural opening behind a depressed fracture of the orbit and the frontal sinus.

Dandy took the fascia from the thigh or from the covering of the temporal muscle. The fascia may be sutured in place or, when this is not practical, laid over the defect.

In August, 1944, Dandy published his work, "Treatment of Rhinorrhea and Otorrhea," presenting eleven cases. Dandy stated that the differential diagnosis can often be made by observing the cerebrospinal outflow:

1. If when the head is tilted forward there is a sudden increase in the volume of fluid, it is apparent that the fluid has been contained in a reservoir and that the fistula is located in a frontal sinus.

2. If the flow of fluid is not altered by tilting the head, an opening in the ethmoid or in the sphenoid cells is indicated.

Dandy's methods by which the fistula may be closed are (1) suturing the dural opening, (2) suturing when possible a transplant of fascia over the dural defect, (3) suturing snugly to the overlying tissues a flap of dura or any soft tissue which has been turned over the bony opening, (4) covering the bony opening with bone wax.

For fistulas through the frontal sinus Dandy devised two methods of approach: one, by elevating the depressed fracture, suturing or covering the defect with fascia, and replacing the depressed fracture; second, if there is no depressed fracture by exposing the frontal region through a unilateral frontal bone flap with a concealed incision, two such procedures are preferable to the single large bilateral exposure, which uncovers and usually requires ligation of the longitudinal sinus.

Dandy states that death following closure of fistula is due to a pre-existing intracranial infection, usually one or more abscesses in the brain.

Eight of the eleven patients whose cases are included in Dandy's report were permanently cured.

Dandy's operation for the treatment of rhinorrhea and otorrhea is practically free of danger.

Restoration of Nerves.—In 1943, Dandy devised a method of restoring nerves, requiring wide resections of neuromas in the upper arm. This is accomplished by resection of the necessary amount of the humerus. It could be applicable in other locations though with aftereffects from the shortening.

Dandy stated in this connection, the following beliefs:

1. The same method can be used when a nerve is resected to cure a tumor along one of the nerve trunks in the upper arm or leg.

2. When a tumor is removed with the nerve, the resection of bone should be done at the same time or within a few days thereafter.

3. It is the only way that function can be restored after an interval that permits larger neuromas to form.

Scaphocephaly.—In September, 1943, Dandy introduced an operation for scaphocephaly. His operative procedure consisted of enlarging the cranial chamber by lifting most of each side of the skull. He fixed the elevated bone flap by a piece of bone cut from the posterior margin of the flap and wired across the defect.

Dandy stated that it is preferable to operate on the right side first, because the major effect of the cerebral protrusion after the dura is opened is borne by the lesser functioning hemisphere.

Intracranial Arterial Aneurysms.—Culminating his brilliant studies of aneurysms, Dandy published in 1944 his book, *Intracranial Arterial Aneurysms,* based on a study of 108 cases with 133 aneurysms. Of this series of cases covering a period of twenty years, sixty-four aneurysms were found at operation and the remaining forty-four were disclosed at necropsy over a period of fifty years.

Up to 1937 no attempt to cure an aneurysm at the circle of Willis by direct attack upon the aneurysm was known in neurosurgical annals. Then Dandy succeeded in the first reported cure of an aneurysm, performing the operation in March, 1937. This was also the first time that an operation had been performed with the diagnosis of an aneurysm beforehand. Since then thirty-six aneurysms diagnosed beforehand (with three exceptions) have been disclosed by Dandy at operation with 70 per cent of cures.

On this basis Dandy concluded that a high degree of accuracy in diagnosing aneurysms can be obtained, and the results are much better than the character of the lesion would have indicated. Dandy demonstrated three requirements:

1. An absolute prerequisite to any form of treatment of an intracranial aneurysm involving the internal carotid or its branches is the knowledge of potential collateral circulation through the anterior cerebral and posterior communicating arteries. This is determined by the Matas test, that is, the obliteration compression of the internal carotid artery in the neck by the finger.

2. If this temporary occlusion cannot be tolerated for ten minutes an attack upon any type of aneurysm would be fatal probably, or at least the patient would be left permanently crippled, for in the vast majority of aneurysms treated surgically the internal carotid must be occluded intracranially.

3. An adequate collateral can be established by partially occluding the internal carotid with a band of fascia lata, reducing the lumen about one-half. Ten days later total occlusion can then be done without risk. In this fundamental book Dandy presents the six methods of operation that he devised: (1) Clipping the neck of the aneurysmal sac; (2) trapping the aneurysm between two intracranial clips; (3) trapping the aneurysm between an intracranial clip and a ligature in the neck; (4) excising the aneurysm and closing the entering vessel; (5) opening the aneurysm and quickly inserting a piece of muscle large enough to fill the sac; the muscle then coagulated with the electrocautery; (6) turning back the aneurysm and coagulating the neck of the sac and the aneurysm itself.

In these operative procedures Dandy used the concealed incision that he devised for the hypohyseal tumors. He believed the following to be true:

1. One of the greatest risks attending any treatment of intracranial carotid aneurysms is injury to the posterior communicating artery.

2. If both the internal carotid and the posterior communicating arteries should be sacrificed, the entire circulation of the brain must be carried from the opposite side through the anterior communicating and anterior cerebral arteries, and this link may or may not be adequate.

3. Simple ligations of the cervical internal carotid may occasionally affect an aneurysm, but is usually ineffective.

4. Without actual exposure of an aneurysm, the diagnosis cannot be established; the carotid ligation may therefore be based in a misconception.

5. Pieces of muscle placed upon or around an aneurysm can scarcely be effective in stopping hemorrhage from a leaking aneurysm, and cannot play any role in curing one. If more direct attack is not possible, the electrocautery is a far better means of closing the point of rupture and offers a chance of cure.

SUMMARY AND CONCLUSIONS

I have mentioned only some of Dandy's outstanding works, because it is impossible in an article of this type to comment on all; there are about 200 publications as a result of his thirty-five consecutive years consecrated to neurosurgery.

Dandy wrote the most brilliant chapter in the history of neurosurgery. The extraordinary merit of his works lies not only in making the most important discoveries in his field, but also in the combined experimental, pathologic, and clinical observations from which the end result—their practical application to surgery—is attained.

To gain a clearer and more detailed idea as to how the evolution of neurosurgery was accomplished through Dandy's works, the years since 1910 may be divided into three periods of ten to fifteen years each:

First Period (1910-1920).—Among Dandy's varied works there are two epoch-making discoveries: (1) The cause, mechanism, and diagnosis of hydrocephalus done in animals and checked on necropsy and the operations for its treatment; (2) cerebral pneumography; both ventricular and spinal injections.

Dandy's discoveries are the foundation in the modern structure of neurosurgery. Without his discovery of the cause of hydrocephalus, the most important subject in neurosurgery, and his complete experimental, pathologic, and clinical studies, neurosurgery would still be far behind its present development. Since its introduction, many papers have appeared in literature suggesting improvements in techniques and a wider application of the principle, but fundamentally little has been added to Dandy's original conception and description. Although each one of Dandy's works constitutes a transcendent landmark in the history of neurosurgery, obviously his discovery of ventriculography in 1918, making possible for the first time the accurate localization of brain tumors, started a new epoch, not only from the technical point of view, but from the intellectual. It transformed radically and integrally the mental conception of neurosurgery, replacing the existent dogmatism and empiricism with a realistic judgment and authentically scientific criterion.

From the historical point of view, if we realize that the three discoveries of the nineteenth century (antisepsis, anesthesia, and cerebral localization) are responsible for the outgrowth of surgery of the brain and that without them surgery of the brain would never be possible, we also realize that without the most important contribution of the twentieth century (Dandy's discovery of ventriculography), surgery of the brain would scarcely be worth while.

Second Period (1920-1935).—In his warfare against neurosurgical ailments Dandy used three different lines of attack, all perfectly synchronized:

1. Since his air injection made possible the diagnosis and precise localization of all brain tumors causing symptoms of intracranial pressure, Dandy revealed the secret abodes of tumors occupying the lateral ventricle and third ventricle and all parts of the brain below the surface. The first tumors of the lateral and third ventricles to be diagnosed beforehand, found at operation and completely removed, were reported by Dandy.

Thus, he radically changed the ill-founded and rather philosophic concept that the normal brain tissue must always be respected and for the first time demonstrated the possibility of removing nonencapsulated brain tumors by extensive resection of contiguous brain tissue, devising the necessary techniques.

2. Dandy introduced his operation for the complete removal of acoustic tumors, giving a scientific explanation of why it had carried the maximum mortality. This constituted the most important landmark in the technical evolution of brain surgery. While developing his operation for the removal of acoustic tumors, Dandy saw the possibility of using the same cerebellar route for introducing his operations for the treatment of Ménière's disease, trigeminal neuralgia, and glossopharyngeal neuralgia, and many fundamental physiologic contributions resulted therefrom.

3. In this third line of attack based on his experimental works, Dandy devised operations for the removal of the hypophyseal tumors and pineal tumors. Using his approach for the hypophyseal tumors, Dandy introduced his transcranial operation for the removal of the orbital tumors.

Third Period (1935-1945).—In this third period Dandy introduced his operations for the treatment of rhinorrhea, scaphocephaly, torticollis, and his method of restoring nerves requiring resection.

In addition to this, Dandy made in this period two more fundamental contributions:

1. During the past few years (1943-1945) he has revolutionized both the diagnosis and treatment of ruptured intervertebral disks introduced by him in 1929.

2. He has done brilliant work on intracranial arterial aneurysms.

The discoverer of ventriculography in 1918, taking the lead in an unexplored field, published in 1944 a book with a complete anatomic, pathologic, and clinical study, on *Intracranial Arterial Aneurysms*, devising the surgical procedures for the treatment and showing a completely new road in the future of neurosurgery.

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COMPLETE EXTRACAPSULAR EXCISION OF TUMORS OF THE HYPOPHYSIS

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THE intranasal approach for tumors of the hypophyseal region has been definitely abandoned by all those whose knowledge is up to date with regard to surgery of the hypophysochiasmatic region. Only unjustified stubbornness would favor this approach today, and it should be formally contraindicated in diseases of this region.

The indication for operating on tumors of the hypophyseal region is given by their influence on the optic tract. Patients are not operated upon to correct endocrine disorders, but to prevent amblyopia and blindness, and also, eventually, to prevent or diminish endocranial hypertension. This can result from a blockage of one or both foramina of Munro, due to the upward growth of the tumor, so that by displacement of the third ventricle an internal hydrocephalus is produced. The majority of the cases which are found in practice, and are the cause of joint collaboration between ophthalmologist and neurosurgeon, have various degrees of atrophy of one or both papillae with more or less marked defects of visual fields. As the compression of the chiasmal region is the principal cause for surgical indication, it is obvious that the liberation of that same compression is the chief object of the operation.

The nasal approach, with a field that is septic and of limited space, passes through the sphenoidal sinus to reach the floor of the sella turcica. Even in the best of circumstances, the surgeon will "bite" the inferior pole of the mass, should this be against the floor of the sella, or most probably traumatize the hypophysis while attempting to excise the tumor, because it is usual for the tumor to displace the gland toward the fundus of the hypophyseal fossa. In a few fortunate patients with cysts (craniopharyngiomas) reached and evacuated through the sphenoidal sinus, there has been later, temporary improvement in the visual field, which explains the enthusiasm of some with regard to this approach. However, this access today should be put aside to constitute only a chapter in the history of surgery on the hypophysis.

The extraordinary improvement in neurosurgery has proved that the intradural approach is the best for surgery on the hypophyseal-chiasmatic region. The extensive incisions, which were originally used by Cushing for extradural access to the sella turcica, have given way to small incisions, practically hidden within the limits of the hair, and with which it is nearly always possible to miss the frontal sinus. The visualization of the operative field is obtained with clarity through a series of technical measures not requiring description here, but which have contributed to reduce the mortality to figures oscillating between 5 and 10 per cent, according to the various clinics and neurosurgeons.

Although the method of choice is the intradural route with a small incision, the surgical manner of treating the tumor itself is a fundamental

chapter and we wish to insist on a few ideas which we consider essential. Very often neurosurgeons are content to reach the tumor, incise its capsule, and lightly curette its contents, leaving the rest of the therapy to x-ray treatment. Without doubt, this is not sufficient. The surgeon should go further, recognizing the extraordinary opportunity he has for obtaining more lasting results, perhaps even definite results, by completely eliminating the mass which is pressing on the optic tracts. It is not exactly the intrasellar portion of the tumor which presses against the optic chiasma, but the supra sellar. While the mass keeps within the sella there is no need to operate, and the surgeon should do so only when the tumor has caused visual disturbance. Usually the tumor also grows behind the chiasma and presses against its posterior aspect. If we should leave this portion of the tumor, and content ourselves with the excision of the anterior or intrasellar portion of it, we would be committing a grave error. Our ideal is to excise the tumor with its capsule. This can be done only in a few cases (Case 1). Usually the capsule of the intrasellar portion of the tumor is intimately adherent to the lining of the sella turcica and this makes its extraction impossible. In these cases, the tumor should be curetted, and should there exist a suprasellar or retrochiasmatic portion, it should be excised. Blind curettage of the tumor frequently causes serious trauma to infundibulum, which is in close contact with the mass. It is necessary to apply traction to the capsule so that it may be dislodged from the posterior aspect of the chiasma and drawn toward the anterior part of the sella, normally occupied by the stalk of the hypophysis, and only then should its contents be curetted and all possible eliminated from the capsule. It is hardly necessary to say that while work is being done close to the hypothalamic centers and the arterial circle of Willis, all this should be done very delicately.

In cases of adenomas, the total extracapsular excision is rarely possible, but may be obtained, as was managed in Case 1. In craniopharyngiomas, total excision is also possible. In many cases, the predominant mass is intrasellar and the capsule and dura intimately fused. In others, the mass has grown chiefly upward, adhering intimately to the infundibulum, and any effort at extracapsular extraction is impossible, because it would doubtlessly produce the death of the patient. Radiotherapy alone can produce acceptable results in the treatment of adenomas of the hypophysis, but is useless in other benign tumors.

X-ray treatment would be justified in adenomas, when the diagnosis for this type of tumor is sure and the optic tracts only slightly altered by the compression, and with the condition that visual field control can be checked periodically. Radiotherapy may cause sudden limitation of visual fields: then the operation should be done without losing time. Today we consider that x-ray treatment should be only the complement of the operation. Its formal indication is in those cases in which, for general reasons, an operation is contraindicated and should be done with all possible care. X-rays are not used, of course, when extracapsular excision has been performed. Postoperative

radiotherapy is indicated routinely in adenomas of the hypophysis, whatever their histology. X-ray treatment is also indicated in small doses in the postoperative treatment of craniopharyngiomas, because it seems to have a certain inhibitory effect on the production of cystic fluid.

CASE REPORTS

CASE 1 (Clinical History No. 20239).—The patient, aged 21 years, was a man from Argentina.

History of Illness.—Five years before admission he had frontal headaches, usually very slight. At times they were strong. During the month before admission he had headaches four to five times a week. He could see little with his right eye for the last two years, and could not read the papers. He was given eyeglasses, but his sight did not improve. During the last two months it became rapidly worse. Asthenia was present and he had little inclination for work.

State of Health at Time of Admission.—His general condition was good, with indifferent decubitus. Nerve I, sense of smell was diminished on the right side.

Fig. 1.

Fig. 2.

Fig. 1 (Case 1).—Dandy's incision of approach to the hypophysis; totally hidden within the limits of the hair. An extracapsular excision of a cystic chromophobe adenoma was performed on this patient.

Fig. 2 (Case 1).—Cystic adenoma of the hypophysis; completely excised.

Nerve II, right eye, showed atrophy of papilla. Left eye, the superior aspect of the papilla was diffuse and veiled, slightly hyperemic. The retina surrounding the superior pole showed the striation of the nerve fiber layer (edema that elevates the internal limiting membrane of the retina) and slight venous traces, bilateral hemianopsia. Nerves III, IV, and VI, extrinsic ocular movement was normal. Nerve V, facial sensibility was preserved. Cranial reflexes were normal. Nerve VII, facial movement was preserved. Nerve VIII, hearing was good. Nerves, IX, X, XI, and XII, nothing unusual.

Active movement was preserved. Passive movement revealed nothing in particular. Sensibility was good; there was no ataxia, dysmetria, nor adiadokocinesis. Reflexes: Patellar, sharp; cutaneous, normal. Psyche was good; standing and walking movements were good.

Clinical diagnosis was tumor of the hypophyseal-chiasmatic region.

Operation.—Operation was carried out,* Dec. 16, 1940, under local anesthesia. Dandy's incision for operation of hypophysis was used, totally hidden within hair limits (Fig. 1). The osteoplastic flap was retracted, and no sign of endocranial hypertension was observed. The chiasmatic region was reached with relative ease. The right optic nerve was found somewhat

*Assistants, Dr. Veppo and Dr. Calzaretto, instrument nurse, Miss Samper.

flattened and in vertical position. Immediately behind it, part of the surface of the tumor could be seen, of reddish-blue color and smooth surface. With bayonet forceps the capsule was perforated and found to be a cyst. Its contents were aspirated and, slowly, traction was applied to the capsule. For some time, we thought it impossible to extract, and considered the possibility of having to sacrifice an optic nerve, but luckily, while continuing the traction, we were surprised to find that the forceps dragged the tumor out whole. It was a cyst of 1½ cm. in diameter (Fig. 2). The width of the wall about 1½ mm. Its anterior surface was smooth and whitish. The hemorrhage which followed extraction of the capsule was only slight and stopped spontaneously. There was never excess pressure applied to the cerebral surface or the infundibulum. Sutured with silk. The bone was fixed with one wire stitch. The patient left the operating room in excellent condition. Pressure maximum 11, minimum 8, pulse 104 per minute.

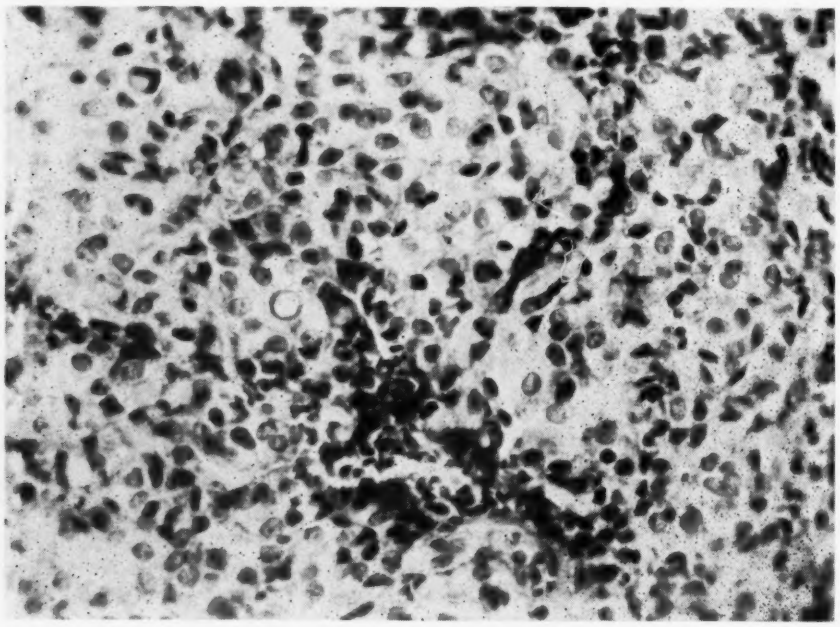

Fig. 3 (Case 1).—Cystic chromophobe adenoma of the hypophysis.

Postoperative.—The postoperative course was uneventful. The patient was discharged on the tenth day after operation. Jan. 17, 1941, one month after the operation, he was again nospitalized with intense headache, vomiting and photophobia, pain in the nape of the neck, temperature of 102° F., and uneasiness. Rigidity of the neck was found. Kernig's sign was positive. For three days he had a sty.

Treatment.—Treatment consisted of suppression of liquid and solid intake, subcutaneous hydration, and sulfapyridine, 3 Gm. A lumbar puncture was performed at 7 P.M., and 40 c.c. of purulent liquid were extracted. The spinal canal was washed with saline solution, and 40 c.c. of antistreptococci serum were injected in this way. Sulfonamide treatment was continued. Jan. 21, 1941, the patient was very much improved and apyrexic. Feeding was started. The patient was discharged on February 1.

Dec. 30, 1942, the patient was well, with the exception of the diplopia due to the palsy of the right rectus inferior. Paralysis appeared after the acute meningitis, one month after the operation. Surgical correction was done.

Aug. 12, 1945, the general condition of the patient was excellent. In spite of the operation performed on his eye, diplopia did not disappear entirely but vision improved. Although hemianopsia was less, the visual field was still defective.

*Pathologic Report.**—Cystic chromophobe adenoma of the hypophysis (Fig. 3).

*By Dr. Pío del Río Ortega.

Case 1 shows the possibility of a complete extracapsular excision of an adenoma of the hypophysis. The late postoperative infection was cured with sulfonamide and antistreptococci serum intraspinally. At that time sulfonamide treatment had just been started in our country. Diplopia was a sequel of meningitis.

Although there was an important improvement in the vision, this case shows that the hemianopsic defect may be permanent if the patient is operated upon late. This was a very rare case of cystic adenoma of the hypophysis.

CASE 2 (Clinical History No. 25052).—A married man, an Argentine aged 42 years, was admitted on April 2, 1943.

History of Illness.—About one year before admission the patient began having frontal headaches in the mornings, localized in the supraorbital region, retro-ocular. After 6 P.M. the pains diminished, disappearing completely at about 9 or 10 P.M. when patient lay in bed. He did not vomit. Simultaneously with the headaches, there was decreased libido, that soon became absolute. Before the onset of the present disease, the patient was hypersexual, and practiced daily coitus. For the four months before admission the patient noticed a decrease of sight in the temporal sides of both visual fields, especially the right. He slept more than normal. His weight was constant. There was no alteration in micturition, in frequency or quantity. His appetite was good.

State of Health at Time of Admission.—His general condition was good. Cranial Nerves: Nerve I, sense of smell was good. Nerve II, the fundus oculi of the left eye was normal. The right eye showed slight atrophy of the papilla. In the visual field there was bitemporal hemianopsia, of the left eye 1, of the right eye 1/10. Nerves II, IV, and VI, good extrinsic ocular movements. Nerve V, facial sensibility was preserved. Cranial reflexes were present. Nerves VII, VIII, IX, X, XI, and XII were without alteration.

Active movements were preserved. Passive movements were normal. Sensibility was good Stereognosia was preserved. There was no ataxia, dysmetria, nor adiadokocinesis. Standing and walking movements were normal. Psyche was without any symptoms.

Probable Diagnosis.—Tumor of the hypophysis.

Operation.—Operation was carried out April 13, 1943.* A right Dandy's incision was used for the hypophyseal approach. There was an abundance of cerebral spinal fluid in the subarachnoid spaces. It was evacuated, facilitating the operation very much. The chiasmatic region was easily reached, and a round, smooth, reddish-gray tumor, emerging like a helmet, was found between both optic nerves. The tumor extended upward, touching the infundibulum. The right optic nerve, which should be horizontal, was nearly vertical. After electrocoagulation of a few vessels, the capsule was cut and an ocherous-colored fluid flowed out. The tumor was cystic. Bit by bit the traction of the capsule gradually drew it forth in various pieces. The breadth of the capsule was between 1 and 2 mm. Once the tumor was extracted, it was found to be situated on top of the diaphragm of the sella turcica, between this, which had been displaced down, and the hypothalamus. It was thought to be a craniopharyngioma. The operation was well tolerated. The bone flap was fixed with a wire stitch. Sutured with silk.

Postoperative.—After the second day, the temperature was high, reaching 104° F., with such excitement, that it was very difficult to keep the patient in bed. He was in a state of fury. For five days he continued the same. At the beginning this state of excitement was not understood, with the possibility of it being a hyperacute infectious process. A lumbar puncture was done, revealing xanthochromic fluid. The bacteriologic examination was negative. After the third day, treatment with sulfathiazole was started, and on the sixth day it was changed to sulfadiazine. Suddenly, this same day, the patient's temperature fell until it reached normal. He was given 18 Gm. of sulfathiazole and 12 Gm. of sulfadiazine.

*Assistants, Dr. Veppo and Dr. Benchimol.

The impression was that the temperature was not influenced by sulfatherapy and was not of infectious origin. The hyperthermia was believed to be of hypothalamic origin. On the tenth day there was again a sudden rise in temperature, to 102° F., which disappeared on the following day. The wound healed by first intention.

*Pathologic Report.**—Glioepithelioma (Fig. 4).

The patient was discharged May 7, 1943. On July 21, 1943, his general condition was good. For the previous fifteen days he had resumed his normal duties. He slept well, but the complete lack of libido continued. Sight was much improved.

December, 1944, at re-examination, visual fields were found normal. Lack of libido continued.

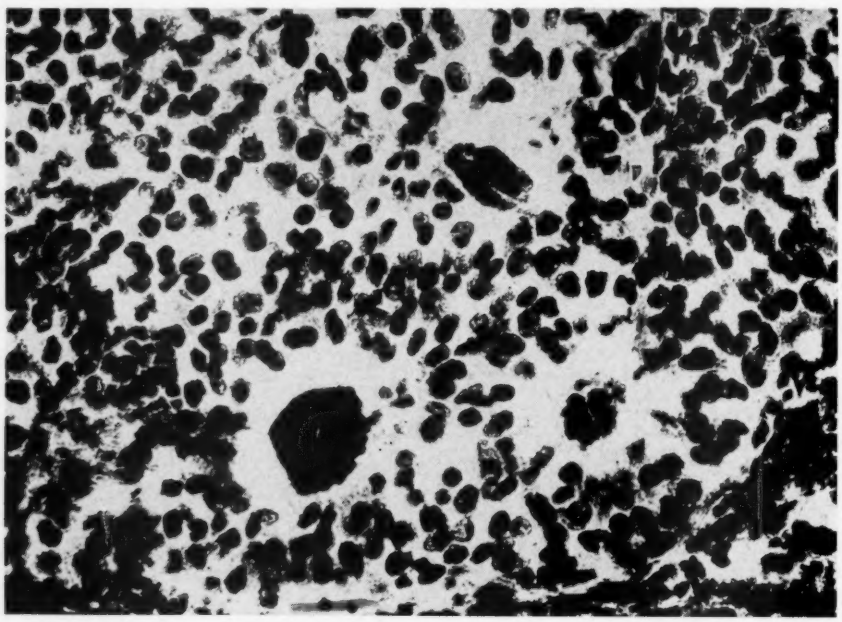

Fig. 4 (Case 2).—Glioepithelioma of the suprasellar region.

This case gives cause for ponderance on some points considered important. Among the symptoms taken in the history was the lack of sexual desire in a man who was previously hypersexual.

Papers by Bard, Dempsey, and Rioch, Brookhart, Day, and Ranson, etc., have made known to us the role played by the hypothalamus in the functional and structural integrity of the genital organs.

Vidal, working with white rats, proved that the destruction of both suprachiasmatic nuclei produces the lack of sexual desire in males. With regard to females, it was principally found that they lacked acceptance of the males.

In this patient (Case 2), the tumor was situated in contact with the anterior part of the infundibulum at the level of the suprachiasmatic zone, which explains the loss of libido, and at the same time corroborates the experimental work of Vidal.

It may be recalled, also, that in the postoperative period there was a state of marked agitation so intense, that at times it was a problem to keep the

*By Dr. M. Polak.

patient in bed. Today, it is accepted without discussion that the lateral hypothalamus exercises a fundamental control in regulating the sympathetic-adrenal system, and plays an important part in emotional conditions, anger, fear, etc.

The right lateral hypothalamus was probably contused in an attempt to excise the tumor, because both adenoma and infundibulum were in close contact. Possibly this is the explanation of the state of fury of our patient, more intense than we had ever seen before.

This picture is wholly different from psychomotor excitement that may follow cerebral operations, and is comparatively common.

SUMMARY

Two cases are presented: one, a cystic chromophobe adenoma of the hypophysis, and the other, a glioepithelioma of the suprasellar portion; both were excised with their capsules following the rules given for this operation by Dr. W. E. Dandy.

The loss of sexual desire in one of the patients must be attributed to lesions of the suprachiasmatic centers of the hypothalamus, whose experimental destruction in white rats, produces loss of sexual desire in males and lack of acceptance of the male by the female.

The great excitement in the postoperative period may be due also to a lesion of the lateral hypothalamus.

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CRANIAL NERVE SURGERY IN THE POSTERIOR FOSSA

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REVIEWING the history of surgery one comes to the conclusion that history of surgery is history of wartimes. The great art of surgery was born on the battlefield and at all times surgery has been trying to keep up with the pace of warfare.

The majority of the great surgical pioneers have known days of war, working in or behind the front lines. Surgeons and soldiers have at all times marched and advanced together, both making quick progress in wartime, both making a tragic and often desperate balance between human destruction and human repair. Saving one young life out of a mass of hopelessly wounded soldiers has always been a human ideal strong enough to arouse the minds of the great surgical pioneers, finding on the battlefield new methods to heal. Tamponade; ligature of the vessels; wound desinfection; treatment of wound gangrene; amputations; splinting of broken bones; transplantation and grafting methods; acute surgery of the cranium, the trachea, the thorax, the abdomen, and the bladder; blood transfusion; and even the development of anesthesia are all counteractions of the surgical pioneers fighting for that tragic balance between human destruction and human repair.

A surgical task which seems absolutely hopeless and useless in peacetime becomes in the next war a routine procedure. As old-time surgery was rooted in the battlefield, so are all the offspring—all date back to times of warfare. Neurosurgery, as the youngest and perhaps most revolutionary descendant, not much older than 40 years, is still old enough to be able to look back on several national wars and on two world wars. Maybe this revolutionary child needed a revolutionary time to grow up and be accepted by the medical profession, breaking with old-fashioned ideas and surgical opinions, and overrunning surgical barriers with the fighting spirit and with the speed of action of a warfaring mind. What seemed impossible was simply done in a new way. Drenched and soiled with the blood of so many young lives, lost in several wars of this so highly explosive and war-bound century, the surgical field became more fertile again and the fertility of the field stimulated anew the minds of the pioneers, bringing on a new surgical era.

This new art of surgery needed indeed new courage and precision, often found in times of war. Surgery of the cranial nerves, particularly in the cerebellopontine angle, is also a real child of this daring century. These children grow up fast and simply do what their parents hardly dared think of. What Jules Verne so passionately dreamed in the nineteenth century, missing the revolutionary technique of the twentieth century, may be compared with the ideas of Charcot, the great member of the French medical academy, who claimed in 1860 that the sectioning of the acoustic nerve in the cerebellopontine angle should stop the attacks of vertigo, described by his great colleague Ménière, as well as with the aspirations of the pathologist Virchow, who traveled all over

Europe in 1900 and visited surgical centers claiming that the intracranial meningiomas could be removed before death came on. The American profession, grounded in the new medical clinics of the twentieth century, accomplished, with great courage and precision, what the old medical world thought hopelessly impossible. Neurosurgery born on the old battlefield of Europe, but not well accepted by the old medical society, needed adoption and education by the daring American profession, working independently with a frontier spirit in newly designed laboratories and in special operating rooms. Once accepted in the country of the great experts, who were courageous enough to risk anything for the benefit of one medical solution, who dared to concentrate themselves on just one small medical sector, this youngest child of surgery has grown up much more quickly and more prosperously than the older ones had predicted.

The medical profession of today is already preparing for the full and widespread development of this new medical art. In another ten years the young neurosurgeons from all over the world will line up in the neurosurgical field to participate in a new competition which will be decisive for the best methods and the best results of this new coming surgical period.

In two brilliant generations the great neurosurgical pioneers have shown the expanse and fertility of the neurosurgical field. Numerous young men in this postwar medical world are now eagerly waiting to take up their new tasks and labors, to divide the field again and give the full energy of their generation, trained and hardened by this second world war, to the never-ending progress of medical science.

Now that I have done neurosurgery independently for more than ten years —now that I have lived and worked for ten years trying to do neurosurgery as I saw it done in the American clinics, particularly in Dr. Walter Dandy's Neurosurgical Department, I think I fully understand the terrific awe that overwhelmed me the first time I saw Dr. Dandy at work in his operating room. Also, I now understand much better the almost painful anxiety that drove me back countless times to his operating room during the three years I visited his clinic in the Johns Hopkins Hospital. More than ever I should now appreciate the moment I could see again this greatest expert of neurosurgical work, who combines in one hand the breath-taking technique of huge bloody brain resections and the fascinating art of bloodless nerve sections in the so vascular region of the cerebellopontine angle.

My admiration for Dr. Dandy continues to increase year by year! Ten years ago I admired Dr. Dandy much more for his work on the gliomas of the brain than I did for those nerve sections in the pontine angle done so easily and quickly. Each time I was due to leave his clinic I tried to postpone my return trip to my home clinic in Holland, not because of nerve sections, but because I knew I should miss tremendously those magnificent surgical moments when Dr. Dandy removed en masse a huge glioma of the brain. I was very sure I could not see enough of these frontal lobe, right temporal lobe, and occipital lobe resections, because these radical procedures showed me exactly how we young surgeons should deal radically with those many malignant

growths in the brain, preferably in the very early stage of their development. This early stage could be reached, as I had seen, by means of Dr. Dandy's ventriculography. I was sure Dr. Dandy showed me the right way to deal with this most difficult surgical problem. It was great to see the master surgeon being confronted with those terrific bleedings and dangerous operative shocks. He was always, with no exception perfectly sure about his surgical technique and knew the value of a fraction of a second. He never gave up, and never missed the tumor, but at the same time he never forgot to estimate, with a perfect feeling, the physical condition of his patients.

And this great and real fighter against these infiltrating gliomas of the brain was never too tired to show me the trail which leads to that medical peak where many satisfactory results in brain surgery are awaiting us.

The good results he had to fight for in late cases, we younger surgeons were to find easily in the future, when the medical profession provided us with early brain tumor cases.

That future has become present time and I am very glad I have followed Dr. Dandy's operative technique as closely as possible. My most radical resections for glioma of the brain have given far better results than have decompression operations, counting an average of twenty radical lobe resections for glioma each year.

In one case (my first) I did successfully a right hemispherectomy. However, this branch of neurosurgery is not the most fascinating one. Dr. Dandy developed also another part of neurosurgery which deals with the nerve sections in the pontine angle. Having practiced this technique for ten years, I now realize to a great extent the quality and quantity of expert work that Dr. Dandy has accomplished in the cerebellopontine angle.

In 1932 to 1933 and 1934 I was with some interruptions, in Dr. Dandy's clinic. I knew his operating room better than my living room on Broadway close to the hospital. Weekly I saw one, two, or even three operations in the pontine angle. It was customary for the daily program to start with a partial section of the sensory root (fifth nerve) at the pons for tic douloureux or the partial section of the eighth nerve (vestibular portion) for Ménière's disease, the second operation being the major one, usually for a brain tumor.

One had to be in the operating room just in time in order to see Dr. Dandy make his unilateral suboccipital approach to the cerebellopontine angle.

The rupture and the profuse bleeding of the mastoid vein was a special point of attraction for the visiting doctors; but many doctors never saw this bleeding because Dr. Dandy placed the bone wax in the mastoid stoma quicker than this big vein could produce hemorrhage. Opening the posterior fossa Dr. Dandy took his time, evading the opening of the mastoid cells, this being a weak point in the aseptic approach to the pontine angle.

The drawing of the curtains in his operating room and the very accurate placing of the headlight by his orderly "Bender" warned the late visiting doctors that the first ten minutes of the operation were over. In the next ten minutes Dr. Dandy did the operation. He opened the dura, then the cisterna

magna and cisterna lateralis to procure sufficient room in the cerebellopontine angle; then he sectioned the nerve (eighth or fifth nerve). In his left hand a small brain spatula (Dr. Dandy's brain spatula) and in his right hand a hooked forceps bayonet-shaped), he slipped one or two wet cotton strips in the pontine angle to protect the cerebellum, asked for the suction and the cautery, and in a few minutes the nerve section was done. In case such a section took a few minutes more, Dr. Dandy used to say "gee whizz, this was a hard one" and we all were sure the work done in these extra minutes would have cost us a good deal more time. Nevertheless, in about six weeks we visiting doctors became so familiar with this operation that we could not understand why there was so much discussion in the American clinics about the problem of the partial section of the sensory root of the fifth nerve. Dr. Dandy's approach was a new, perfect method which could be done safely in twenty to thirty minutes, as we had seen in his own clinic. The discussion on the treatment of Ménière's disease seemed finished to us too! The operative treatment was so simple and could be done so quickly, as we had seen, that this section of the vestibular portion had to be considered in the future as the treatment of choice, even for the pseudo-Ménière cases.

In one case, I remember quite well, it took Dr. Dandy one full hour to complete a difficult tic operation. There seemed to be a number of vessels in his way. Several of these vessels had to be thrombosed with the cautery. However everything progressed smoothly. At the end of the operation Dr. Dandy turned round and said: "If you ever run into a case like this one, Dr. Verbeek, you had better close and take the subtemporal route."

This was the first time I felt somewhat uneasy about the suboccipital route to the fifth nerve.

In Dr. Dandy's clinic I saw no serious postoperative complications either in a case of tic or in a case of Ménière's disease in a combined series of 100 operations. In two patients the wound had to be reopened, the patient having developed high temperature and high cell count; accumulation of fluid was found in the operative field. Drainage restored their condition.

Twice in three years I saw a beginning of a keratitis following total resection of the sensory root. The eyelids of these two patients were sutured and the keratitis disappeared readily. These patients were told to wear a special eye-shield for some time. I did not see a facial weakness nor a motor root paresis, and after the *partial* section of the sensory root of the fifth nerve there was still some preservation of sensation for light touch, deep pressure, temperature, and sharp and dull, particularly in the region of the first and second branches. In some cases the cornea reflex was preserved; in other cases this reflex was very much diminished, depending apparently on anatomic variations in the pontine angle (accessory fibers for the cornea reflex).

After having seen some twenty operations for tic and about half as many for Ménière's disease, I thought I was ready to go home. So far as these operations were concerned, I had studied every detail of them; I had made plenty of drawings and taken notes in the operating room; I knew every little movement

Dr. Dandy made during these procedures; I could not miss finding and sectioning these nerves. I was sure these operations would not get on my nerves.

Following Dr. Dandy's advice I went to Philadelphia in the early spring of 1932 to visit Dr. Charles Frazier's clinic. It was a very interesting and enjoyable time I spent in this clinic as in a short time one felt oneself a member of the staff "family," which meant also a member of the Frazier family, Dr. Frazier being a kind father to all of us. Very soon I understood Dr. Frazier was doing a great deal of work—particularly many tic operations following the subtemporal route.

The first operation I saw done was Tic operation No. 690. Dr. Frazier was an expert and his tic patients did very well after the operation. As a rule the patients were out of bed on the fifth day, and left the hospital on the eighth.

Having seen an expert, as Dr. Frazier was, I hesitated to voice objections against his method, comparing this tic operation with Dr. Dandy's method. In a less experienced hand the results of this operation would certainly be less excellent. The following objections might be presented:

1. There was a total anesthesia in the trigeminal area, sensory fibers of which were sectioned behind the gasserian ganglion. The best and most physiologic operation for tic douloureux is the operation that cuts only the pain fibers. (The patients of Dr. Olof Sjöqvist, who performed a tractotomy, had total analgesia of the three branches but also an absence of cornea reflex and thermhypesthesia.)

2. Some of these patients complained very strongly about numbness.

3. The tic patients with pain in the region of the first branch had to have an alcohol injection or a section of the supraorbital nerve.

4. As a whole this subtemporal operation seemed to me more complicated. In each case one had to control the severe arterial bleeding of the middle meningeal artery and the profuse venous bleedings around the gasserian ganglion close to the foramen ovale.

5. Liberating and dividing the sensory root and saving the motor root impressed me as a method requiring long-standing experience.

6. Dr. Frazier told me that in an average of one out of fifty cases he had to do the operation in two stages because of the profuse bleeding, and Dr. Frazier was an expert.

I left Dr. Frazier's clinic still preferring Dr. Dandy's method, but very happy to know the subtemporal route should there be too many big vessels in my way in the cerebello pontine angle. In the summer and fall of 1932 I visited Dr. Harvey Cushing's clinic, the Mayo Clinic, and Dr. Max Peet's clinic. In all these great neurosurgical centers the subtemporal route was considered safer and was used routinely. On my short visits I did not see an operation for Ménière's disease. The subtemporal operation for tic douloureux was in these clinics in very safe hands.

On a second visit to the clinic of Dr. Frazier I was very much astonished to hear Dr. Frazier tell me, "And now, Doctor, I'm going to show you what a poor neurosurgeon I am." I did not know what to say, but I certainly protested.

Dr. Frazier showed me, in the ward, a woman who had a right-sided keratitis, a right-sided trigeminal anesthesia, a right facial paralysis, a complete right-sided deafness, a dysarthria and a marked right-sided ataxia. "Well doctor, what is your diagnosis?" Dr. Frazier asked. Having visited so many good neurosurgical clinics, I did not hesitate in telling Dr. Frazier I thought this woman had a right acoustic tumor in a very far stage of development. Dr. Frazier answered very dryly that this was his first tic case in which he had followed Dr. Dandy's suboccipital route. This was the second time I felt uneasy, and this time very uneasy, about Dr. Dandy's tic operation. I realized that special neurosurgical operations had to be done by special neurosurgical experts.

Dr. Harvey Cushing told me this when talking about the hypophyseal operations. In the winter of 1932-1933 I was back again in Dr. Dandy's clinic, not only to study his radical brain resections for infiltrating gliomas, but also to make myself much better acquainted and, if possible, quite familiar with this wonderful approach to the fifth and eighth nerves.

In any case, I had to master this approach in order to be able to do the operation for Ménière's disease. In 1933 I did not go back to Holland, but remained in the United States until the end of 1934. In these wonderful years spent in Dr. Dandy's clinic, I saw the most radical operations for brain tumors I could dream of and so many operations for tic douloureux and Ménière's disease that I was convinced again that these operations could be done safely. To be exact, I knew of no safer intracranial operation as long as these nerve sections of the fifth, eighth, and ninth nerves were in Dr. Dandy's hands. In 1941 I received a letter from Dr. Dandy telling me he had just lost his first patient with Ménière's disease. It was No. 358. The patient died from a postoperative meningitis. I do not know of any better results in a series of this magnitude. In Dr. Dandy's clinic I saw three patients with neuralgia of the glossopharyngeal nerve. The ninth nerve was sectioned in the cerebellopontine angle, with very good results. In one case the patient proved to have accessory fibers of the ninth nerve, which had to be sectioned afterward, as the patient had a recurrence of this tic douloureux. In between the ninth and the tenth nerve two accessory fibers were found and sectioned. Following this second operation the patient remained completely free from pain.

Writing about cranial nerve surgery in the pontine angle, I should like to express my hope that some day Dr. Dandy will write a book on the removal of the eighth nerve tumors (the encapsulated tumors of the cerebellopontine angle), as Dr. Cushing did for his pupils in 1918. The operative treatment of these benign, well-encapsulated but dangerously located tumors, I consider as the best test for a man's neurosurgical capacities. I know of no more difficult task a surgeon can face. Dr. Dandy practiced the most radical removal of these tumors. In several other clinics the intracapsular excochleation was advocated. Dr. Dandy himself considered the total removal of these tumors as the fiercest battle in the neurosurgical field. Still he chose this battle every time he had to remove such a tumor. I am convinced that only a master surgeon like Dr. Dandy can afford to be in favor of this radical procedure in late cases. Particularly

in a late case, where the tumor has become strongly adherent to the brain stem, I doubt whether this ultraradical removal of the mesial side of the tumor capsule can be advocated as the method of choice for the average neurosurgeon.

Following the thrombosing or clipping of the vessels, which cross from the brain stem to this mesial side of the tumor capsule, the postoperative edema of the brain stem is a complication seen too frequently. I am afraid the mortality would be prohibitive in a long series of these cases. In an early case, there is no doubt, this radical procedure must be the method of choice. Maybe we are approaching the day when the general medical profession is going to recognize this tumor syndrome of the cerebellopontine angle more frequently. A unilateral diminishing of hearing, with or without tinnitus, accompanied with a weakened homolateral cornea reflex or the patient's complaint that dust is always getting into the same eye (same side), will then make more doctors suspicious of the development of a tumor in the cerebellopontine angle.

The unilateral approach to the cerebellopontine tumors, as advised by Dr. Dandy in September, 1934, is, I think, far superior to the bilateral suboccipital craniectomy as generally used for the removal of these tumors. The latter method is not only a far too formidable procedure, taking too much of the patient's already weakened resources, but a unilateral approach provides all the room there is need for. Moreover, the unilateral approach prevents a great deal of mishap, which may occur (in each case of high intracranial pressure) with the wide opening of the posterior fossa. The very dangerous shifting of the midbrain toward the incisura tentorii and the protrusion of the cerebellum through the widely opened posterior fossa, causing hemorrhages and postoperative edema of the midbrain and cerebellum, make the operation unnecessarily hazardous even before the tumor can be attacked.

This protrusion and swelling of the cerebellum and brain stem are also apt to cause a mental change of the patient, not rarely ending in a postoperative delirium, followed again by more protrusion and edema. Likewise, the edema of the cerebellum and brain stem causes poor respiration and poor swallowing, very frequently leading to a postoperative pneumonia. The coughing and difficult expectoration again cause progressive edema of the protruding cerebellum, rapidly closing this disastrous circle. A clear-cut intracranial tumor syndrome deserves a clear-cut and direct route to the tumor, saving a great deal of operative risk. The tapping of the lateral ventricles, the opening of the cysterna magna in order to release as much fluid as possible, the excision of the protruding cap of the cerebellum are all three essential for this unilateral approach. They safely provide all the room there is in the cerebellopontine angle and make the bilateral suboccipital craniectomy entirely unnecessary.

On one point I did not follow Dr. Dandy's technique. Dr. Dandy is in favor of the use of avertin anesthesia with a small additional dose of ether. This anesthesia is perfect, as long as an expert anesthetist* is administering the ether. In Holland I terribly missed an expert anesthetist, particularly for

*Miss G. Smith worked with Dr. Dandy over thirteen years.

these cerebellar operations. Finally, I had to apply local anesthesia. I must say I became accustomed to it and even like it now. In those cases where I needed an additional general anesthetic I tried, one year ago for the first time, 1 c.c. scophedal, injecting this 1 c.c. solution slowly into the transverse sinus. The patient, as a rule, goes to sleep within ten minutes and keeps quiet during the whole operation (one and one-half to two hours). There is no venous congestion in the cranium and the tumor in the pontine angle can be approached much more easily. How much I wish I could forget all the troubles and mishaps I met, particularly in approach to the cerebellopontine angle during my first years of neurosurgical work in Holland.

Returning to Holland in the early days of 1935 I was very happy to see again, after three years, the old windmills, the dreamy and quiet canals with their old-fashioned sailing boats, and the old dykes, built decades and even centuries ago, but I was frightened when I saw also the old-fashioned operating room where I had had my first years of surgical training in the University Hospital of Groningen. Formerly I was happy in that operating room, working day and night, hoping later to be able to do any operation, no matter how difficult and dangerous. Just like all the assistants I wanted to become an all-round surgeon and in this large University Hospital, where the surgical department was always overfilled with all kinds of surgical patients, I knew I had found the best place for this surgical training.

Educated for general surgery, I left this so beloved operating room in order to study neurosurgery in America. Returning after three years I did not want any longer to become an all-round surgeon.

In the latter the art of surgery becomes so diffused that it loses the sharp and fine character of expert work.

I had been looking forward to a neurosurgical operating room, which, as I soon learned, was not to be built. I tried very hard to feel at home again in this old general surgical operating room. It was the heart of the clinic and I endeavored to get back my old love of this place. All the general surgical work of the University Hospital of Groningen was done in this room. We worked on two tables, when we were rushed with work. This room was also the old-style lecture room or theater for the students, and last but not least this room was used for dressing the wounds of very ill patients.

This operating room with a huge window could not be darkened. My proposal of having curtains in the operating room was not accepted. I knew I would have difficulty doing an operation on a deep-seated tumor or doing a nerve section in the cerebellopontine angle. Nevertheless, when neurosurgical patients were brought to the clinic, I felt I should at least try to do my work, mainly because these patients had no better place to go in this part of the country.

I was so lucky as to remove a left frontal parasagittal meningioma successfully; I operated upon a patient for a pneumocephalus with good result; further, a medullary tumor and also a cystic glioma in the left cerebellar hemisphere were removed in the first two months. These two patients developed bad wound infections, but at length they improved.

Then came my first tic case—a woman suffering from multiple sclerosis and a severe tic douloureux of all three branches of the right trigeminal nerve. Following Dr. Dandy's method I made a right suboccipital craniectomy, the patient being under avertin ether anesthesia. Exploring the cerebellopontine angle, I could just see the eighth nerve but I could hardly distinguish the sensory root of the fifth nerve because the use of the headlight was of very

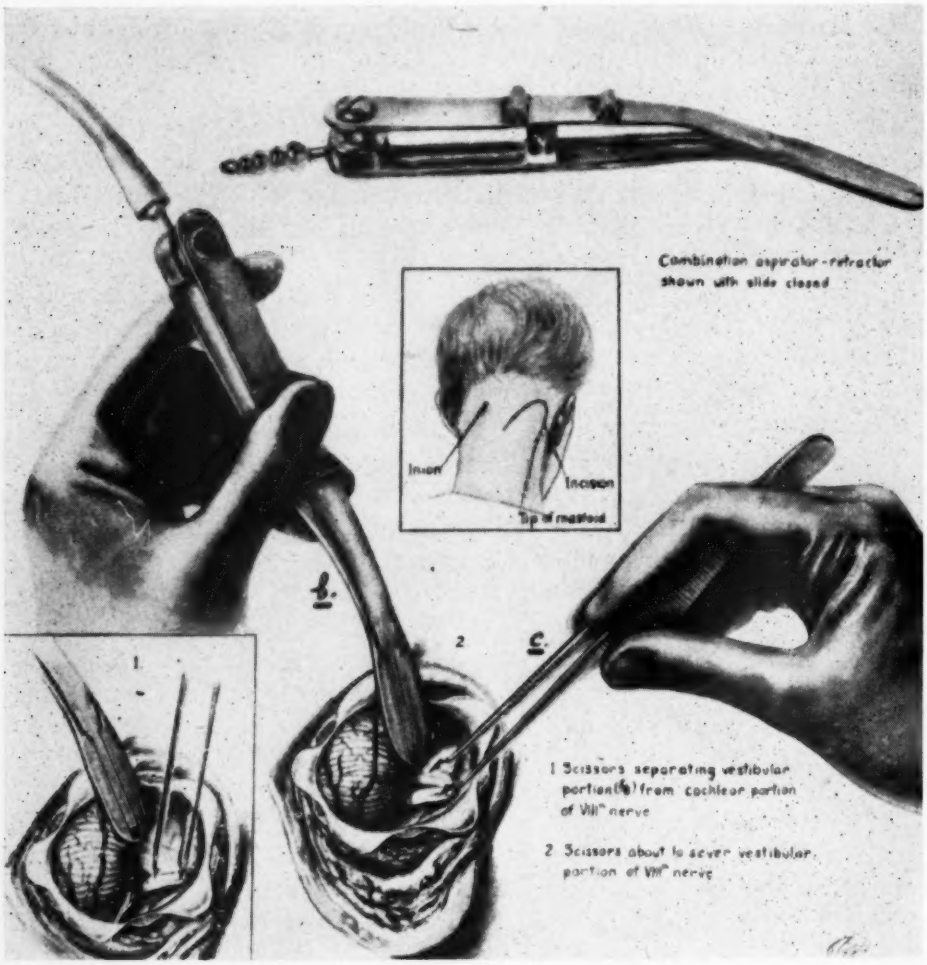
Combination aspirator-retractor shown with slide closed
Incision
Incision
Tip of mastoid
1
2
1 Scissors separating vestibular portion (8) from cochlear portion of VIII nerve
2 Scissors about to sever vestibular portion of VIII nerve

Fig. 1.

Figs. 1 and 2.—Showing the method of using the instruments designed by the author. (These two drawings were made in the School of Artists, connected with the Johns Hopkins Hospital, Baltimore. The original drawing of the operative field was made by Miss Dorcas Hager in 1931; the copied drawing, with the instruments, was made by Mr. Osgood in 1934.)

little help in an operating room that could not be darkened. Moreover, the anesthesia had to be stopped because the patient became very cyanotic. We did not have a special anesthetist for brain operations.

I worked hard for three hours. The patient survived but I had to wait twenty-four hours before she responded again. I am happy to say her recovery was further uneventful and the patient left the hospital within three weeks,

completely free from pain, the wound well healed, and no sign of keratitis nor facial weakness.

I had the same trouble operating on my second tic patient. The anesthesia had to be stopped again, there being much bulging of the cerebellum. Exploring the cerebellopontine angle, I could see the sensory root only very faintly, because the headlight had a very weak focus in this operating room, which still could not be darkened. This operation lasted two hours forty-five minutes. The patient

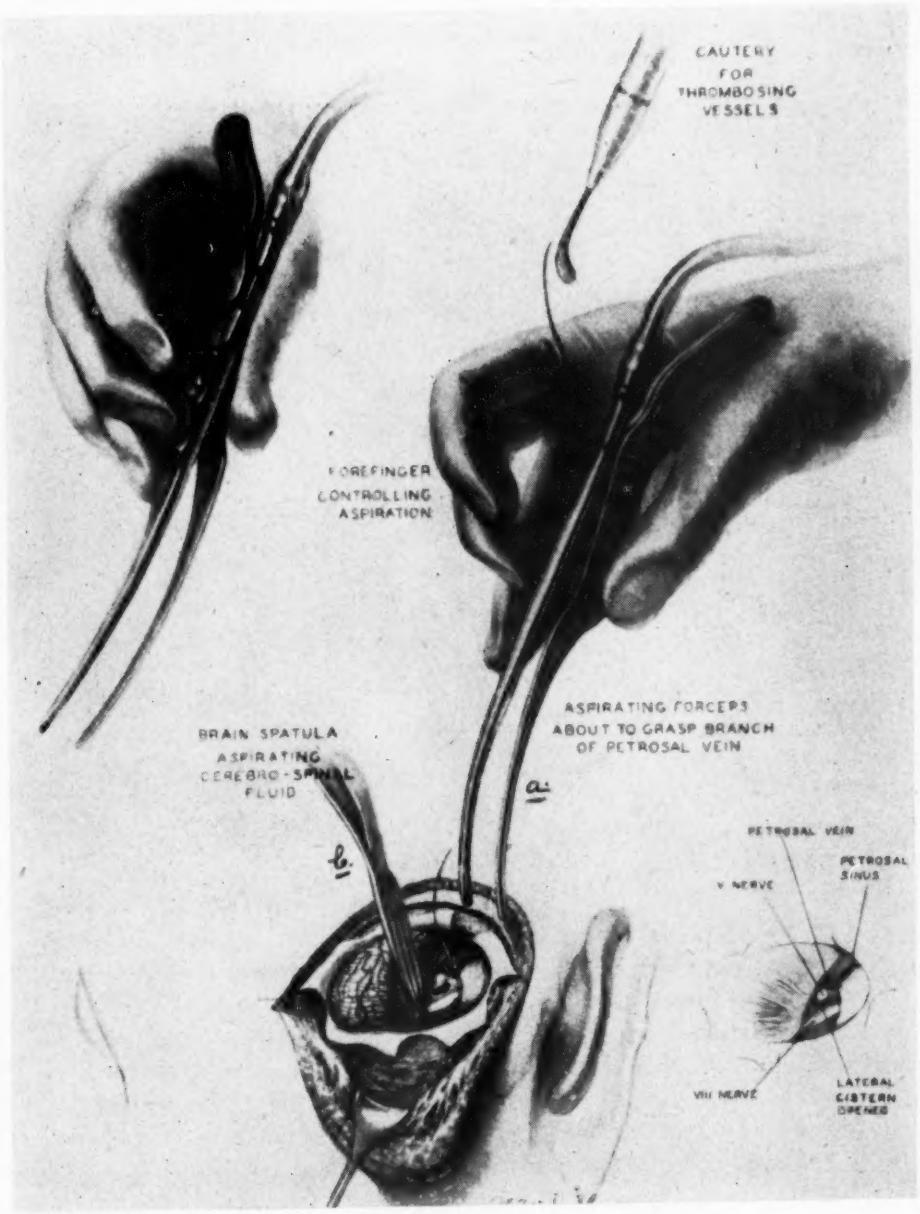

Fig. 2.—*(For legend see opposite page.)*

did well and was free from pain. Nevertheless, I decided not to operate again on the cerebellopontine angle as long as the operating room was not adapted to this type of work. I endeavored to improve the conditions, did several operations for brain tumors, medullary tumors, meningoceles, and so on, but drifted slowly back to general surgical work. The hospital was to be rebuilt but was not to have a special neurosurgical operating room.

I tried to forget my grievances doing a great deal of general surgical work. If I had not remained in America so long I should certainly have given up neurosurgery. By this time I felt so attached to this specialized work that I could stay away from it no longer.

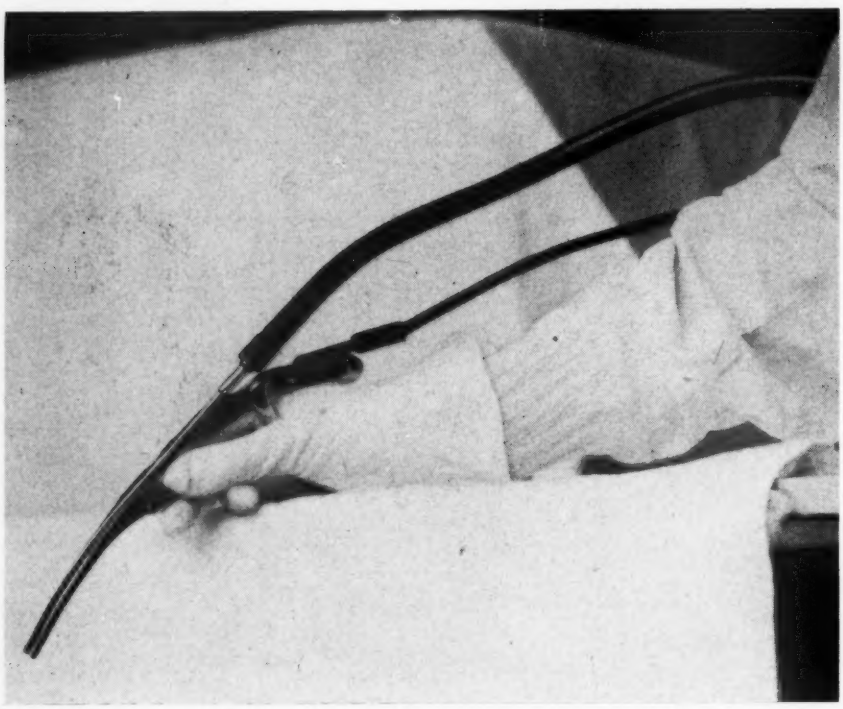

Fig. 3.—The photograph shows the forceps, connected both with the suction apparatus and with the electrocautery.

By the end of 1936 I felt I had done enough general surgery and I decided to start my neurosurgical work independently.

The first thing I asked for when coming to the private Catholic hospital of Gromingen was a special neurosurgical operating room and a special staff of nurses. In a few years the number of patients increased so much, that the Catholic Sisters decided to have also a special neurosurgical department in the hospital with a very modern operating room, which was to be used exclusively for neurosurgical work. In July, 1941, this new department was opened and the work has been increasing very rapidly.

Particularly for the work in the cerebellopontine angle and also for the operations on deep-seated tumors, I have been using two instruments which I designed when I was studying in the Johns Hopkins Hospital.

One instrument is a brain spatula or retractor, which can be used as a suction apparatus. The other instrument is a bayonet-shaped forceps, which is not only connected with the suction, but also with the electrocautery apparatus (see Figs. 1 to 3). Controlling a bleeding in a deep-seated place, I use the brain-retractor with my left hand, exposing the bleeding point and aspirating the blood and the cerebrospinal fluid. At the same time I place the forceps on the bleeding point. This point is then kept dry, because the blood and the fluid are continuously aspirated through a small opening in one leg of the forceps, carrying the suction tube. This forceps is also continuously connected with the electrocautery apparatus. The bleeding point is therefore automatically coagulated, as soon as the forceps is placed on it. The switch for the electrocautery is on the operating table. Having a sudden and severe bleeding from a deep-seated point in a narrow operative field one really does not need much help. The only thing that has to be done is to expose the bleeding point with this brain retractor and place this forceps on the bleeding point; the rest is done automaticallyy. In ten years I have never used a silver clip to control bleeding. Particularly in the cerebellopontine angle, I feel much safer when using these two instruments.

I am perfectly sure Dr. Dandy will smile faintly at the idea of these instruments. He is never worried about any bleeding and, moreover, he knows very well how to avoid bad bleedings, particularly in the cerebellopontine angle. When a beginner wants to examine the sensory root of the fifth nerve, all the vessels in this angle seem to be in the way and several of them are apt to get torn before the sensory root can be seen.

The cerebellopontine angle is really not a location for a beginner. It is a gorge full of pitfalls.

I have done over eighty operations on the cerebellopontine angle. Still I never forget to use my two semiautomatic instruments. I have now done a series of forty operations for trigeminal neuralgia, following Dr. Dandy's method. In another series of ninety-cases I used Dr. Frazier's approach. Now that I have tried both methods I am sure I prefer Dr. Dandy's. It is a more physiologic operation. One is able to cut the pain fibers of all three divisions, preserving a good deal of sensation in the first and second divisions. The absence of the cornea reflex is even less frequent than after the tractotomy (Sjöqvist operation).

To be sure one has to be very familiar with all the technical details of this operation. I had to wait a long time before I could do this operation safely.

DISCUSSION

1. The anesthesia is, I think, the main technical point of this operation. If the anesthesia is not perfect, there will be too much bulging of the cerebellum. In such a case it is hardly justifiable to undertake a partial section of the sensory root of the fifth nerve at the pons. I now use local anesthesia (pantocain 1 0/00) with 1 c.c. scophedal, injected into the transverse sinus during the operation.

2. The cerebrospinal fluid must be drained very slowly from the cysterna magna and lateralis cysterna in order to prevent vomiting.

3. The petrosal vein must be thrombosed and divided before the sensory root is exposed. This can be done easily and safely with the help of my instruments.

4. The arterial loops covering the sensory root should, if possible, be carefully held aside. Cauterization is rather dangerous because these loops are too close to the brain stem.

5. This partial section can be done safely only in a dark operating room, the operator using a very good headlight.

6. The dura defect must be closed, either by suturing back the small dura flap or by using a fascia muscle transplant as Dr. Dandy has advised.

In some cases I still prefer Dr. Frazier's operation, particularly for patients with a very short and fat neck, and also for patients who are very old (75 years of age and over) I think Dr. Frazier's method is a safer procedure.

In the Dandy series I lost one patient (79 years old) who developed a postoperative pneumonia. The operation was done under ether anesthesia. Another patient, 62 years old, who had a heart failure, died suddenly on the second postoperative day, complaining much about precordial pain.

In the Frazier series I lost also two patients. A woman of 66 years died in postoperative shock; a woman of 71 years developed a bronchopneumonia. This woman was also suffering from multiple sclerosis.

The intracranial operation I like best of all is the partial section of the eighth nerve for ménière's disease. I have now performed twenty-seven of these operations. All the patients are doing well. This partial section of the eighth nerve can be done much more easily than the section of the fifth. The petrosal vein should be left untouched and one hardly needs to retract the cerebellum. As a rule I use only local anesthesia without scophedal. Before sectioning the vestibular portion I warn the patient that an attack of dizziness may be coming, which does not always happen. An occasional vomiting does not disturb this operation. Under local anesthesia there is hardly any bulging of the cerebellum, even when the patient starts vomiting. When the patient is subtotally deaf on one side, I divide the eighth nerve completely, hoping to cure the patient also from the annoying tinnitus. I wish I could start the daily program every day with an operation for Ménière's disease. In the last twelve days I did four operations for tic and three for Ménière's disease. I operated on two glossopharyngeal tic patients with very good results. Patients with a benign encapsulated tumor in the cerebellopontine angle are not rare in this country. Unfortunately, the majority of them come to the neurosurgeon in the very last stage of tumor development. Therefore I have not practiced the most radical and total removal of these tumors yet. I operated on twenty-one patients. I made a unilateral suboccipital approach, resected a small overlying cerebellar cap, incised the tumor capsule, and curetted as much intracapsular tumor tissue as possible. Then I removed as much of the tumor capsule as possible, leaving the mesial side attached to the brain stem. Except for one, these patients did fairly well after the operation. One patient died three weeks after

the operation from a bronchopneumonia caused by difficulty in swallowing. In two patients the signs of intracranial pressure disappeared only partially. I operated a second time and extended the suboccipital craniectomy bilaterally with good results. In the next twenty years I hope to operate very radically on these benign tumors in an early stage of their development.

SUMMARY

1. The development of intracranial surgery, particularly in the cerebellopontine angle, is briefly reviewed.

2. The operations for lesions of the cranial nerves in the cerebellopontine angle are discussed.

3. A series of 130 operations for major trigeminal neuralgia is reviewed.

4. In a series of forty cases Dr. Dandy's suboccipital route and in a series of ninety cases Dr. Frazier's subtemporal route was followed.

5. As a result of these 130 operations I prefer Dr. Dandy's operation.

6. The technical points of this operation are stressed.

7. Two new instruments for intracranial surgery are introduced.

8. The section of the eighth nerve for Ménière's disease, following Dr. Dandy's method, is reviewed in twenty-eight cases.

9. The operative treatment of the benign encapsulated tumors in the cerebellopontine angle (acoustic tumors) is discussed and twenty-one cases reported.

10. The section of the ninth nerve for glossopharyngeal tic done by myself in two cases is described.

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THE REPAIR OF CRANIAL DEFECTS BY BONE GRAFTING

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INTRODUCTION

AS LARGE numbers of persons received wounds of the head during World War II, often with extensive loss of bone and damage to the brain, the time is now opportune to review the position regarding the repair of cranial defects and take stock of past results. The loss of portion of the cranial vault from trauma or other cause has always been regarded as likely to have serious consequences. Even if hernia cerebri does not occur, there may be some bulging of the scalp and meninges from an underlying collection of fluid, and pulsations, synchronous with the heartbeat, are usually visible and palpable. Becoming more marked with effort and with lowering of the head, both these conditions become rather terrifying to the patient, and objects of interest to relatives and friends. Provided the dura mater has not been damaged, there is, however, no great likelihood of dangerous sequelae, and restoration of the skull is necessary only for cosmetic reasons or for protection of the brain against further injury. When the dura mater has been injured, or portion of it destroyed, then the loss of bone is more serious and may allow adhesions to occur between the brain, damaged meninges, and scalp, causing severe symptoms and real disability. Should the brain have been damaged also, the resultant gliosis and brain scar become adherent to the scalp and a traction diverticulum forms from the lateral ventricle of the same side. Epileptiform fits of jacksonian type are then likely to occur, as well as lose of function, corresponding to the site of the trauma.

Even if the brain itself has not been injured, changes in character and behavior, headaches, fits, etc., seem to be expected of any person who has a "piece of bone missing from his head," and all sorts of dire consequences are supposed to follow a blow on the unprotected area of brain. The patient lives in dread of these consequences and rapidly develops an inferiority complex. Various forms of protective metal and leather pads are often worn under the headgear. An excuse is present for misconduct, and even criminal tendencies, and also for shortcomings and chronic invalidism as far as his earning capacity is concerned. Thus, in the absence of specific contraindications, repair of the cranial vault and damaged meninges should be carried out, not only for the relief of symptoms and signs caused by adhesions and brain scars, but also for psychologic and cosmetic reasons, and to obviate the wearing of external protections, such as jockey's caps with metal inlays, etc.

MATERIALS

The question of the most suitable material for repair of these defects has always been a vexatious one. An excellent review of the subject and the various

substances in use was given by Grant and Norcross[1] (1939). In the earliest case on record, in 1670, bone from a dog was successfully used. Over 200 years passed before any further reports of plastic operations on the skull appeared. Then followed a thirty-year period during which various types of bone grafting procedures, including the use of the outer table of the skull, were advocated. In an endeavor to avoid two operations upon the one patient, plates made from various inorganic substances were then tested with varying degrees of success. Celluloid came first and continues to be employed by many surgeons. Then followed aluminum, gold, and silver. Results with inorganic materials were, on the whole, unsatisfactory to the surgeon and patient chiefly because of sepsis; and to the lay public they never rendered the patient immune from aftereffects. Indeed, the possessor of a "plate in the head" was always regarded with morbid curiosity, and was expected to become epileptic or insane. In Case 18 of this series, where celluloid had been inserted some ten years previously, a cyst had formed between layers and was the cause of severe symptoms.

Autogenous osteoperiosteal or cartilage grafts became popular again during World War I. They were usually obtained from neighboring outer table of the skull and transferred to fill in the missing area by various ingenious maneuvers. In other cases, free grafts were fashioned from tibia, split ribs, sternum, and scapula. However, these bones were not satisfactory donor sites because it was necessary to use several pieces to fill in large defects; a compromise was effected by the use of soupbone or boiled cadaver skull bone which could be cut to any size or shape. Indeed Nafziger[2] (1936) advocated that bone flaps affected by osteomyelitis and intrinsic tumors should be boiled and then replaced to obviate unsightly deformities and dangerous defects.

In 1931, Pickerill,[3] advocated the use of the inner table of the ilium bone; in 1932, I[4] reported two patients treated by this method. The pendulum has now swung again toward inorganic materials. Robertson[5] (1944), using plates from a new metallic element, tantalum, claims to have found the theoretically ideal plastic material which is chemically and electrically inert in the tissues.

In 1944, Kazanjian and Holmes[6] reported thirty-eight patients in whom reconstruction of the frontal bone had been carried out after radical operation for osteomyelitis. When large grafts were needed, these authors had used vitallium strips and plates, tantalum plates, and, in one instance, an acrylic mold. They admitted that inorganic substances were unsatisfactory, because even the newer materials which were allegedly inert caused foreign body reactions in the tissues and had the psychologic disadvantages mentioned previously. They regarded living bone as the ideal material, but because they favored the tibia or the crest and anterior portion of the ilium as donor sites for strips of bone, they only advocated bone grafting for areas less than two and one-half inches in diameter. They were apparently not aware of the method of obtaining large grafts from the inner table of the ilium, which is regarded as the ideal graft for the skull for the following reasons:

1. A large enough portion can be obtained in a single graft to fill in almost any deficiency.

2. It is curved in the correct planes and can be molded further if necessary.

3. Its inner surface is smooth and not likely to become adherent to the underlying dura, even where it has been repaired or replaced by a graft of fascia.

4. Its outer surface of cancellous bone invites a fresh blood supply from the overlying scalp, to keep the graft alive.

OPERATIVE TECHNIQUE

The pathologic lesion in the head must be dealt with first. The scalp, whether it be intact or composed largely of scar tissue, must be reflected well beyond the limits of the missing bone, and the dura or its remains must be identified. Where the dura and brain have been damaged, scar tissue and cerebral gliosis should be excised as far as practicable until healthy cerebrum or the ependyma of the ventricle is encountered. The dura must be freed beyond the edge of the defect in the bone and then reconstituted with the aid of a graft of fascia or other substitute, if necessary. A bed for the graft is prepared by removing any irregular or indriven pieces of bone and beveling the edges of the gap; a pattern is made the exact size of the gap from a piece of zinc foil.

For the cutting of the graft an incision is made just above the crest of the ilium, as described by Pickerill[3] (1931). The abdominal muscles and fascia are detached with a knife close to the bone. The periosteum is divided along the inner aspect of the crest, and the iliacus muscle is stripped from the inner table of the bone. It is then retracted medially with a wide-bladed Royle retractor, until sufficient area is exposed to accommodate the malleable metal pattern. This is placed in position with its longest border along the iliac crest and a graft of corresponding size and shape is marked out with a sharp instrument. It is most important to cut through the inner table along these lines with a narrow osteotome first, before separating up the graft, otherwise the field becomes obscured with blood and the limits of the graft cannot be seen. A wide chisel, with a slight degree of flexibility or curvature, has been found most suitable for cutting through the diploe evenly. Bleeding from the ilium bone is controlled with Horsley's wax, and the wound is then closed with a soft drain which is left in for thirty-six hours. The graft itself can be trimmed to fit into its bed accurately with strong scissors. In the earlier cases of the series, it was simply fixed in place with absorbable catgut sutures, but since the availability of stainless steel wire, it has been wired into place. The scalp is replaced and the wound closed accurately. Drainage for thirty-six to forty-eight hours is advisable and a large protective dressing should be applied.

INDICATIONS

Some of the indications for cranioplasty have already been discussed and along with the remainder, they may now be summarized as follows:

(1) Post-traumatic fits associated with missing bone, adhesions, a cerebral scar, and traction diverticulum of a ventricle

(2) Headaches, and the syndrome of the "defective cranial vault," namely, dizziness, lack of concentration, loss of energy, mental instability, and intolerance of noise, etc.

TABLE I. SYNOPSIS OF CASES

CASE NO.	SEX	AGE AT OPERATION (YR.)	DURATION, CAUSE, NATURE AND SITE OF INJURY, AND TREATMENT ELSEWHERE	DURATION, SYMPTOMS, AND SIGNS	ENCEPHALOGRAPHY	DATE AND NATURE OF OPERATION, AND TYPE AND SIZE OF GRAFT	IMMEDIATE RESULT	FOLLOW-UP RESULT	CATEGORY
1	M	8	1 yr.; knocked down by motorcycle; compound comminuted depressed fracture with laceration of brain; right parietal; immediate craniectomy	1 yr.; slight hernia cerebri; no symptoms; wore jockey's metal skull cap	Not done	September, 1931; repaired dura not disturbed; ilium graft 4 cm. in diameter	Primary healing	Slight headaches when studying at age 15; killed in action in Army, New Guinea, 1942	Satisfactory
2	M	37	35 yr.; cause unknown; compound depressed fracture and laceration of brain; right parietal; immediate craniectomy	16 yr.; nocturnal fits following blow at site of missing bone; 7 yr.; second craniectomy without relief	Cortical atrophy with meningeal adhesions and cyst formation; no diverticulum	November, 1931; excision of subarachnoid cyst, cerebral scar, and adherent meninges; dura repaired with Cargile membrane (Fig. 1) May, 1932; large graft of ilium 8 by 3 cm.; full thickness in center	Primary healing; some relief by third operation; fracture of ilium one month after fourth operation during fit	Occasional headache; no fits for 8 yr.; no sedatives; continuous work as cleaner; some absorption of graft, January, 1945 (Fig. 2)	Satisfactory
3	M	14	2 yr.; kick from horse; compound comminuted depressed fracture and laceration of brain; left frontal; immediate craniectomy	2 yr.; hernia cerebri; no symptoms; cosmetic defect	Not done	April, 1934; meningeal cyst evacuated; ilium graft 6 by 3½ cm.	Primary healing	Occasional headache; has been 4 yr. in Army (anti-aircraft battery) January, 1945	Satisfactory
4	M	20	2½ yr.; motor car accident; depressed fracture with middle meningeal hemorrhage; left temporal; immediate craniectomy	2 yr.; jacksonian fits; once a month average	No definite abnormality of ventricles or cortex	December, 1934; dura mater adherent to cortex; ilium graft 5 by 3½ cm.	Primary healing of skull wound; infection of ilium wound from wax; sinus for 1 yr.	Approximately 2 fits every year, but at continuous work on docks and in railway workshops	Relieved

5	M	23	8 mo.; blow with hammer falling 120 feet; compound comminuted depressed fractures involving eye and orbit, and laceration of brain; left frontal and left orbit; immediate repair	6 mo.; headaches; one fit; serious cosmetic defects; left eyeball in antrum; orbital margin and roof inside cranium; unable to wear glass eye (Figs. 3 & 4)	Not done; plain roentgenograms show extensive damage (Figs. 5 & 6)	August, 1935; removal of some bone fragments; severe meningeal hemorrhage; ilium graft 10 by 5 cm.	Subarachnoid hemorrhage; primary healing; glass eye fitted	Two fits in next three years; no headaches, well since, good appearance; working hard as timber cutter, January, 1945	Satisfactory
6	M	6	1 yr.; knocked down by motor car; compound depressed fracture and laceration of brain; left parietal; immediate repair	No symptoms; cosmetic defect	Not done	July, 1937; ilium graft 3.5 by 3.5 cm.	Primary healing of skull wound; infection of ilium wound from wax	Good cosmetic result; not traced since November, 1937	Untraced (?satisfactory)
7	M	27	15 yr.; cerebral abscess after influenza; right frontal; craniectomy and drainage	13½ yr.; hernia cerebri; fits, once a month average	Large diverticulum of anterior horn of right lateral ventricle	July, 1937; separation of scalp adherent to brain scar; ilium graft 5 cm. in diameter; March, 1939; excision of brain scar; diverticulum decapped; graft reinserted	Primary healing, then recurrence of fits; bony union found between graft and skull at second operation	Fits less in frequency; developed diabetes, sustained compression fracture of thoracic spine; died of diabetes, July, 1942	Relieved
8	M	29	7 yr.; motor car accident; compound depressed fracture; right parietal; immediate craniectomy	6 yr.; jacksonian fits every two weeks (average); hemiplegia, worse since second operation elsewhere 8 mo. before	Large diverticulum from temporal horn of right lateral ventricle	August, 1937; foreign body (gauze swab) removed and adhesions freed; March, 1940; cadaver bone graft; 5 by 3 cm. (Fig. 7)	Primary healing on each occasion; no relief after third operation	No sedatives; no fits since 1940, left hemiplegia much improved, but crippled with rheumatism; roentgenogram shows graft almost intact (Fig. 8) January, 1945	Satisfactory

TABLE I.—CONT'D

CASE NO.	SEX	AGE AT OPERATION (YR.)	DURATION, CAUSE, NATURE AND SITE OF INJURY, AND TREATMENT ELSEWHERE	DURATION, SYMPTOMS, AND SIGNS	ENCEPHALOGRAPHY	DATE AND NATURE OF OPERATION, AND TYPE AND SIZE OF GRAFT	IMMEDIATE RESULT	FOLLOW-UP RESULT	CATEGORY
9	M	7	6 mo.; knocked down by motor car; compound comminuted depressed fracture and laceration of brain; right frontal; after immediate operation, hernia and abscess	3 mo.; meningoencephalocoele, and cosmetic defect; slight headaches	Large irregular diverticulum from right lateral ventricle	April, 1938; excision of brain scar and chronic encapsulated abscess; outer table of large fragment used as graft (Fig. 9)	Primary healing	No symptoms; good cosmetic appearance; no absorption of graft (Fig. 10), January, 1945	Satisfactory
10	M	25	11 yr.; fall from horse; compound comminuted depressed fracture and laceration of brain; right frontal; immediate repair	1½ yr.; headaches, severe fits in bouts after exertion; cosmetic defect	Large regular diverticulum from right lateral ventricle, still present in December, 1944 (Fig. 11)	November, 1938; excision of depressed fragments, dirt, and brain scar, diverticulum decapped; cadaver graft 9 by 6 cm. (Fig. 12); December, 1944; removal of remains of graft (Fig. 13); ilium graft 9 by 6 cm. (Fig. 14)	Transient aerocele; primary healing	No fits, no headaches; enlisted A.I.F. in 1939; fits recurred in the Middle East, 1941, and increased to December, 1944; too soon after ilium graft for prognosis	Unsatisfactory
11	M	13	9 mo.; hit with golf ball when caddying; comminuted depressed fracture; dura penetrated; right temporal; immediate craniectomy	No symptoms; slight hernia cerebri	Early dilatation of right lateral ventricle; no diverticulum	January, 1939; dura not opened; ilium graft, 5 cm. in diameter	Primary healing of skull wound; ilium wound infected due to wax	No symptoms; working hard on sheep station, January, 1945	Satisfactory

12	M	26	7 yr.; motor car accident; compound comminuted depressed fracture; hemiplegia; right frontal and temporal; immediate craniectomy	5½ yr.; headaches; jacksonian fits once a month average	Generalized dilatation of right ventricle; no diverticulum	April, 1939; adhesions separated; cadaver graft 9 by 6 cm.	Primary healing	No fits for one year; recurrence on ceasing sedation, in bouts every six mo., in service 3 yr., continuous work as dental mechanic; in February, 1945, graft completely absorbed (Fig. 15)	Unsatisfactory Regrafting indicated
13	M	18	9 yr.; blow from sheet of iron; compound comminuted depressed fracture and laceration of brain; left parietal; immediate craniectomy	2 yr.; fits, once a week average	Large diverticulum from body of left lateral ventricle	June, 1939; excision of cyst and brain scar; diverticulum decapped; ilium graft 9 by 5 cm.	Reactionary hemorrhage of both wounds; infection of ilium wound (no wax used)	"Reasonably free of fits; may go for six months without one"; working on farm, January, 1945	Relieved
14	M	23	1 yr.; motorcycle accident; compound comminuted fracture with laceration of brain; right frontal; immediate craniectomy	3 mo.; headaches; one fit	No diverticulum and normal cortical pathways	December, 1939; scarred dura not disturbed; cadaver graft 6 by 4 cm.	Primary healing	Had one fit five months after operation, nil since, worked very hard, no sedatives, roentgenogram shows some absorption of graft (Fig. 16) but clinically firm skull, February, 1945	Satisfactory
15	M	18	8 yr.; fall off horse; compound comminuted fracture with indriven fragment of bone; right occipital; wound repair only	6 mo.; frequent fits, fields of vision defect; no relief by operation for removal of depressed fragments of bone	Localized cortical atrophy with diverticulum from posterior horn of right lateral ventricle	January, 1940; excision of brain scar and wide decapping of ventricle; cadaver graft 6 by 3.5 cm.	Reactionary hemorrhage; infection, ependymitis, and meningitis; removal of graft; sulfonamides; death	Nil	Unsatisfactory (death)

TABLE I.—CONT'D

CASE NO.	SEX	AGE AT OPERATION (YR.)	DURATION, CAUSE, NATURE AND SITE OF INJURY, AND TREATMENT ELSEWHERE	DURATION, SYMPTOMS, AND SIGNS	ENCEPHALOGRAPHY	DATE AND NATURE OF OPERATION, AND TYPE AND SIZE OF GRAFT	IMMEDIATE RESULT	FOLLOW-UP RESULT	CATEGORY
16	M	13	1 yr.; diving into shallow water; depressed fracture; right parietal; immediate craniectomy	No symptoms except neurosis	No abnormality seen	February, 1940; dura intact; ilium graft 7.5 by 5 cm.	Primary healing	No symptoms; enlisted in Army, 1944	Satisfactory
17	M	24	6 yr.; hit on head with piece of wood in brawl; comminuted depressed fracture; left parietal; immediate operation and postoperative hemorrhage	2½ yr.; headaches; right hemiplegia; generalized fits progressing to status epilepticus; bad cosmetic defect with deep gutter in scalp	Large diverticulum from left lateral ventricle; scarred cortex 1 cm. in depth	May, 1940, partial excision of scar; decapping diverticulum; repair of dura; ilium graft 6 by 4 cm.; plastic on scalp	Primary healing	No headaches, no fits since September, 1941; good cosmetic result; no sedatives; residual paresis of right hand; continuous work, bone graft intact (Fig. 17), February, 1945	Satisfactory
18	F	55	35 yr.; as a child; undefined injury, right frontal; craniectomy for adhesions, 5 years; removal of cyst from brain; celluloid graft inserted	4 yr.; severe throbbing frontal and occipital headaches; neurosis; tender depressed scar; consistently high intracranial pressure of 220 mm.	Slight compression of anterior part of right lateral ventricle; cortical atrophy	February, 1944; removal of two plates of celluloid and "cyst" between them; evacuation of shallow cyst deep to false dura; ilium graft 3 cm. in diameter	Primary healing, after evacuation hematoma	Immediate relief of severe headaches and throbbing sensation; traced for only four months	Relieved

19	M	18	13 yr.; fall 20 feet off balcony; compound comminuted depressed fracture, cerebral laceration, and hemorrhage; right temporal; immediate operation	5 yr.; jacksonian fits; worse with service in R.A.A.F.; homonymous quadrantic defects in visual fields	Huge diverticulum from right lateral ventricle to reach site of irregular and depressed fracture; scarred cortex only few millimeters in depth (Figs. 18 & 19)	May, 1944; excision of displaced fragment of bone and cerebral scar; ependyma of left ventricle intact; dura repaired with fascia from external oblique muscle; ilium graft 4 by 3 cm.	Primary healing; some reactionary hemorrhage from ilium wound	No major fits; three minor uncinate "turns" but nil since, on light sedation; graft intact after nine months (Fig. 20); continuous work, February, 1945	Satisfactory
20	M	26	6 yr.; motor car accident; depressed fracture and cerebral laceration; right parietal; two operations	6 yr.; headaches; left hemiplegia, with severe degree of flexor contracture of left hand and forearm; 2 yr., fits since fall from mast of ship, increasing to status epilepticus	Large smooth diverticulum from anterior horn and body of right ventricle; brain scar 2 cm. in depth	August, 1944; removal large irregular fragment; excision of brain scar; dura repaired with fascia lata; outer table of fragment replaced as graft	Primary healing	Without sedatives, fits recurred, but none for three months on sedation; remarkable improvement in hemiplegia and use of left hand, February, 1945	Relieved

Note: During the interval between Cases 17 and 18, the author was on full-time military service with the Australian Imperial Force.

from three months to sixteen years after their onset, for the following reasons: fits, 13 cases; headaches, 8 cases; hernia cerebri, 4 cases; hemiplegia, 3 cases; neurosis, 2 cases; cosmetic defects, 2 cases; protection, 2 cases.

In 16 cases, the dura mater and/or brain had been damaged. In 10 of them, encephalography demonstrated the presence of a cerebral cicatrix with traction diverticulum for the underlying lateral ventricle. In all these cases, excision of the cicatrix with its surrounding area of gliosis was performed as far as was practicable (without producing further loss of function) and the dura mater reconstituted, prior to the fixation of the graft.

In 3 cases, the repairs and the grafting were done at separate operations, and in 1 patient (Case 10) a second graft had to be inserted where cadaver bone had been absorbed. The inner table of the ilium bone was used 14 times, cadaver skull 5 times, and autogenous skull 2 times. In 3 cases, free grafts of fascia were needed to repair the dura mater.

Fig. 2 (Case 2).—Lateral roentgenogram taken in February, 1945, nearly thirteen years after the first graft (Fig. 1), showing a good deal of irregular absorption of the ilium graft.

DISCUSSION

Some of the cases presented are worthy of special comment, especially Case 5. This patient sustained an injury in the left frontal region and orbit, when a heavy hammer fell 120 feet down the shaft of a mine in which he was working. Primary operation and débridement were performed at a country hospital, and a very satisfactory recovery took place, considering the severity of the trauma. When seen about eight months later, however, the patient complained of headaches and some slight disability with speech and urinary con-

(3) Feelings of discomfort, pulsation, and throbbing at the site of the defect, usually associated with cerebral hernia and changes of weather

(4) The need for protection against further injury to the brain, associated with certain occupations and sports

(5) Unsightly cosmetic defects causing an inferiority complex

CONTRAINDICATIONS

With the advent of the sulfonamides and penicillin, there are now few local contraindications for cranioplasty; but, if the original wound has been infected, and if there remain indriven bone fragments and foreign bodies, a period of at least three months from the time the wound has healed should be allowed to elapse before grafting is attempted.

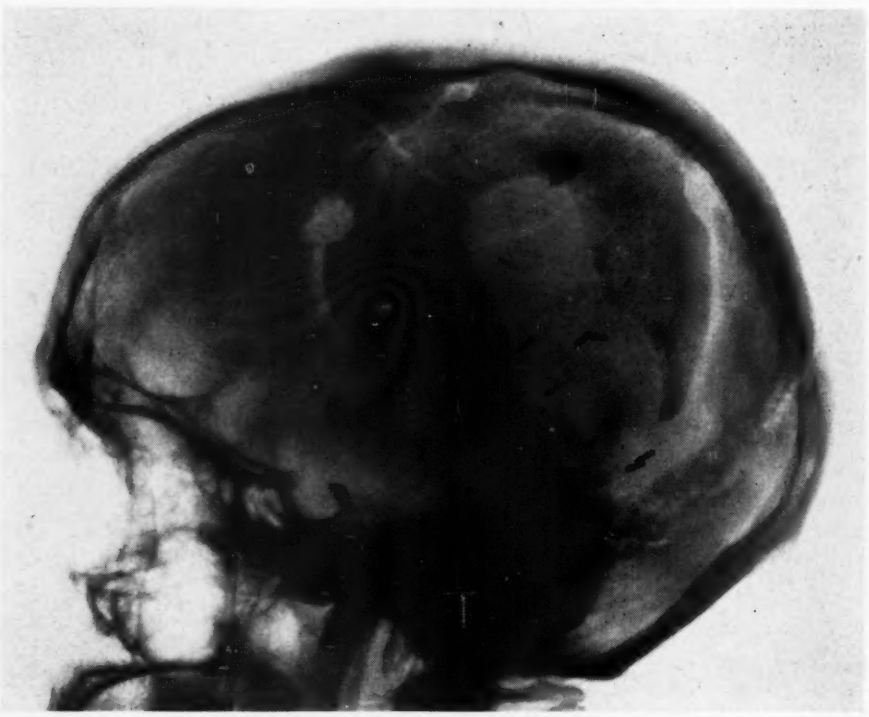

Fig. 1 (Case 2).—Lateral roentgenogram taken before the insertion of the ilium graft in May, 1932.

ANALYSIS OF CASES

In support of the foregoing contentions, a series of 20 consecutive cases with 21 grafts is submitted. Details of these cases are shown in Table I. As might be expected, there were 19 men and only 1 woman. Excluding the woman their ages varied from 6 to 37 years, the average being 20 years. The various regions of the skull were affected as follows: frontal, 8 cases; parietal, 8 cases; temporal, 3 cases; occipital, 1 case.

The onset of symptoms and signs for which relief was sought varied from three months to nineteen years after the trauma; operation was undertaken

trol; he had had one major epileptic fit. He presented an extraordinarily bad cosmetic appearance (Figs. 3 and 4). He could not voluntarily open the left eyelids, but on separating them the eyeball was seen displaced downward into the antrum. The extent of the damage to the skull and orbit and the indriven fragments can be seen in the roentgenograms (Figs. 5 and 6).

At the operation, alarming extradural hemorrhage was encountered when removal of one of these fragments was attempted. It probably came from the sphenoparietal sinus, and was controlled temporarily by packing with gauze. Following a parlous convalescence, a satisfactory cosmetic result was obtained. Only two fits occurred during the next three years; in a letter written in December, 1944, he stated he was symptom free and working strenuously as an axman in a pine forest.

Fig. 3. Fig. 4.

Fig. 3 (Case 5).—Front view, showing the deformity of the left frontal region and orbit.
Fig. 4 (Case 5).—View of the left side.

Another patient (Case 10), in addition to fits, also had a bad cosmetic result after injury. His appearance was almost identical with that of the patient shown in Fig. 3 of the article by Grant and Norcross[1] (1939). Following removal of several indriven fragments of bone, foreign bodies (dirt), and an extensive area of gliosis, the gap in his skull was filled in with a large piece of bone, removed some time before from a patient with an inoperable brain tumor (Fig. 12). No further fits or headaches occurred, and two years later he enlisted in the Royal Australian Engineers A.I.F., sailing for the Middle East in

Fig. 6.

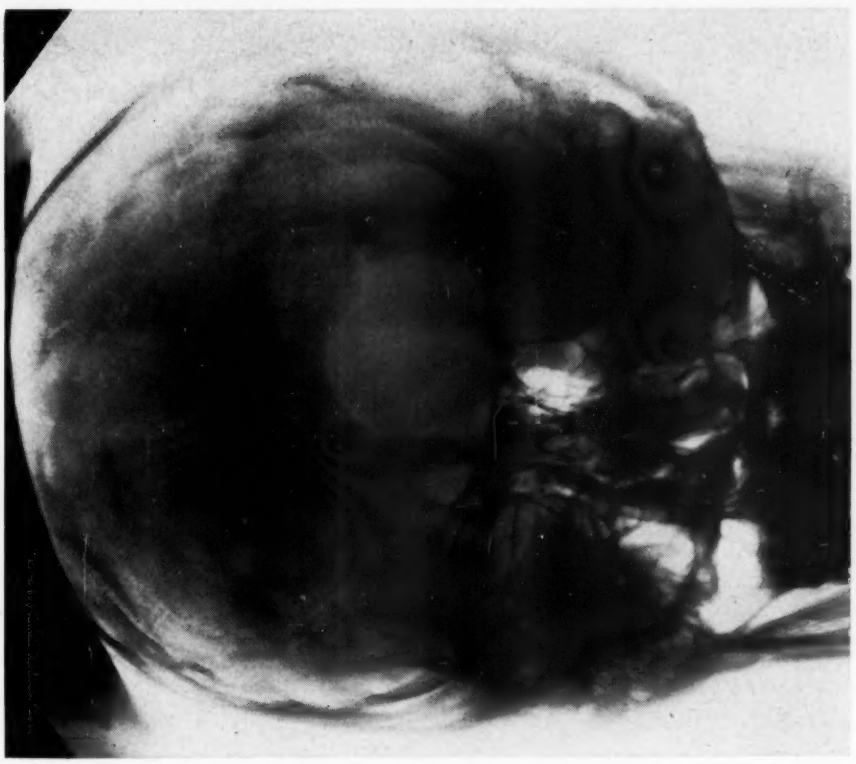

Fig. 5.

Fig. 5 (Case 5).—Anteroposterior roentgenogram showing area of missing bone and indriven fragments.
Fig. 6 (Case 5).—Lateral view of the facial bones showing the left eye inside the left antrum.

Fig. 7 (Case 8).—Lateral roentgenogram showing the large cadaver graft, April, 1940, shortly after its insertion.

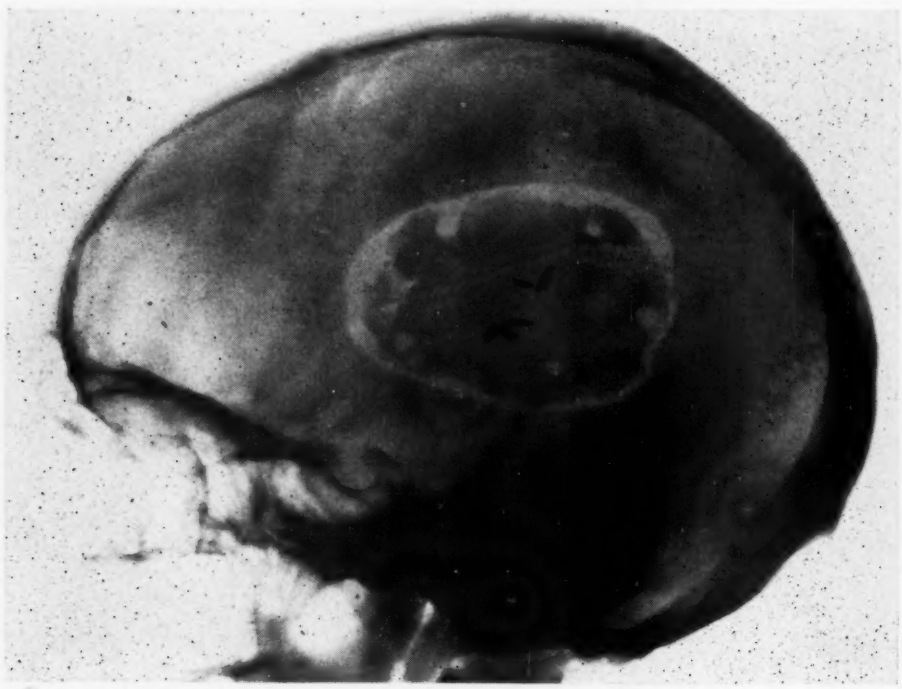

Fig. 8 (Case 8).—Lateral roentgenogram taken in December, 1944, showing only a very slight amount of absorption after nearly five years.

Fig. 9 (Case 9).—Lateral roentgenogram taken in March, 1940, two years after the insertion of the outer table of a large fragment of the skull as a graft in April, 1938.

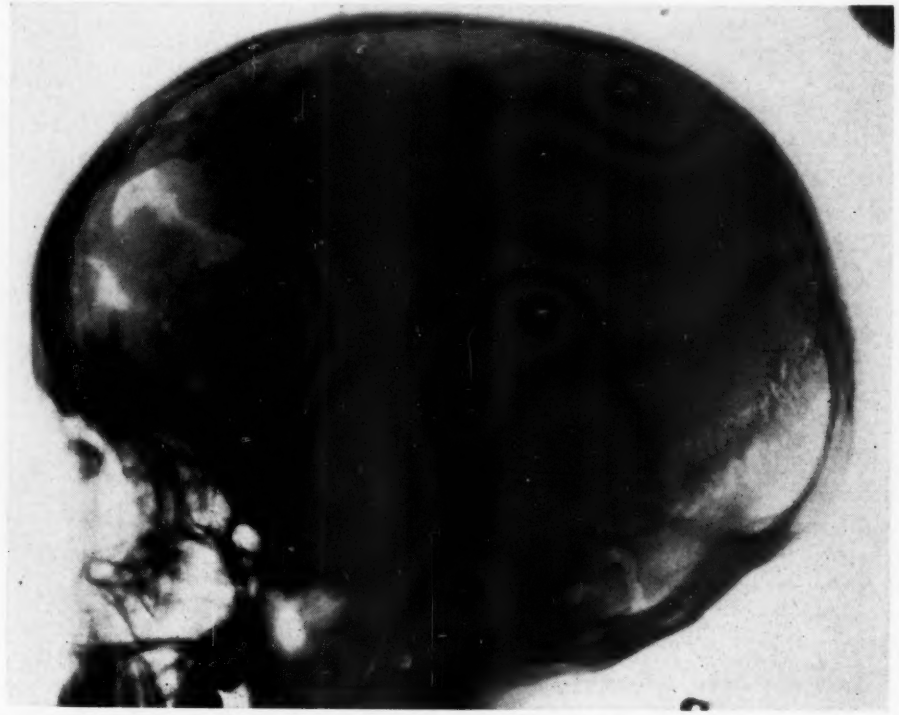

Fig. 10 (Case 9).—Lateral roentgenogram taken in December, 1944, showing that there has been practically no absorption of the outer table graft, and that bony union has occurred with the skull in places.

Fig. 11 (Case 10).—Encephalogram taken in December, 1944, showing the large diverticulum from the anterior horn of the right lateral ventricle, which re-formed after the operation in 1938.

Fig. 12 (Case 10).—Lateral roentgenogram showing the cadaver graft in position shortly after its insertion in November, 1938.

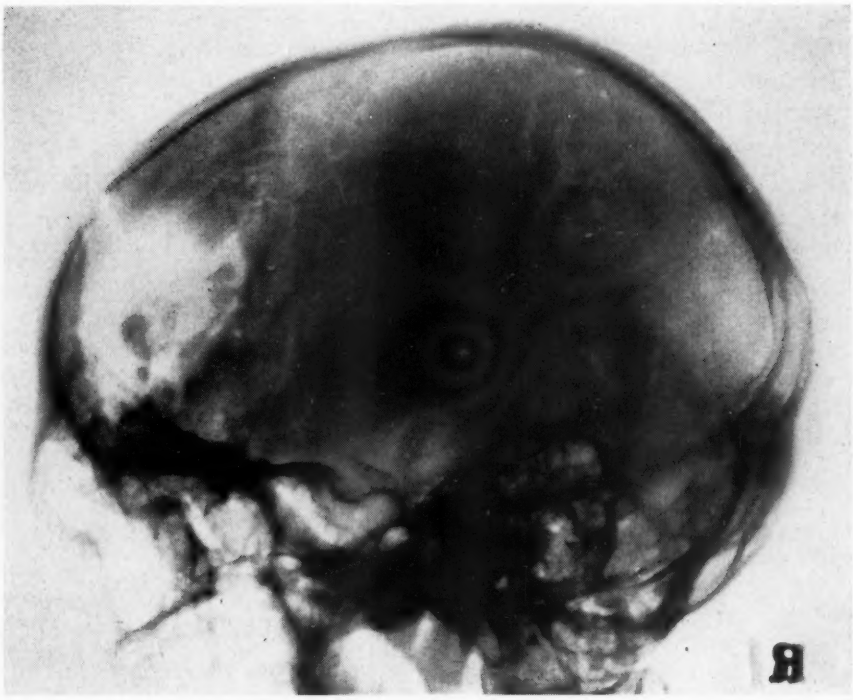

Fig. 13 (Case 10).—Lateral roentgenogram taken in December, 1944, showing extensive absorption of the cadaver graft after six years.

Fig. 14 (Case 10).—Lateral roentgenogram showing the ilium graft wired in position, January, 1945.

March, 1940. After taking part in the first Libyan Campaign in 1941, the fits recurred with gradually increasing frequency and severity; he was repatriated and discharged from the Army in 1942. When he was re-examined in December, 1944, the fits were occurring about three times a week. The grafted area felt very irregular and was tender. A roentgenogram (Fig. 13) showed that practically the whole of the graft had been absorbed. The traction diverticulum from the right lateral ventricle was still demonstrable by encephalogram (Fig. 11). The remains of the cadaver graft were removed, together with some additional fragments of displaced bone, and a graft from the ilium was wired in place (Fig. 14). It is too early to predict the final outcome but while the patient was under sedation no fits had occurred for one month after operation.

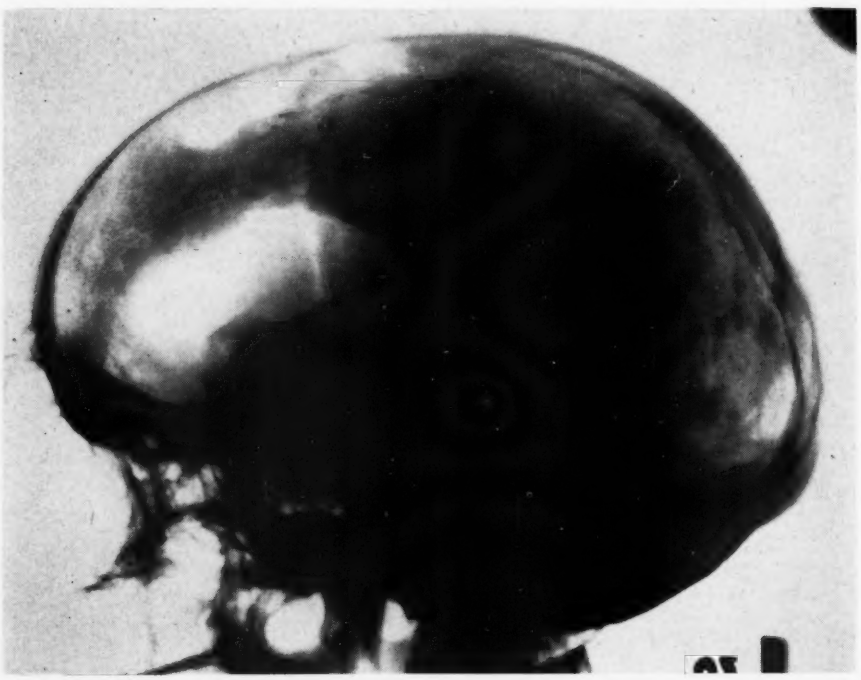

Fig. 15 (Case 12).—Latural roentgenogram showing complete absorption of a cadaver graft inserted nearly six years previously. The roentgenogram taken shortly after the insertion of the graft has been lost.

Case 18 (the only woman patient in the series) was of interest because of the length of time the headaches had been present. The nature of the original injury which occurred in childhood could not be determined nor the pathology (if any) for which the first operation was done in the country thirty-five years before. Two celluloid grafts had been inserted at a second operation, elsewhere, six years before, but headaches had recurred within a few months. Subsequently, some regeneration or overgrowth of bone occurred around the edges of the gap in the skull, and at the third operation, one year before, a collection of fluid, enclosed in a smooth adventitious sac, was discovered between the two layers of celluloid. Removal of the celluloid plates, the "cyst," and the new

Fig. 16 (Case 14).—Lateral roentgenogram showing only patchy absorption of cadaver graft inserted five years previously.

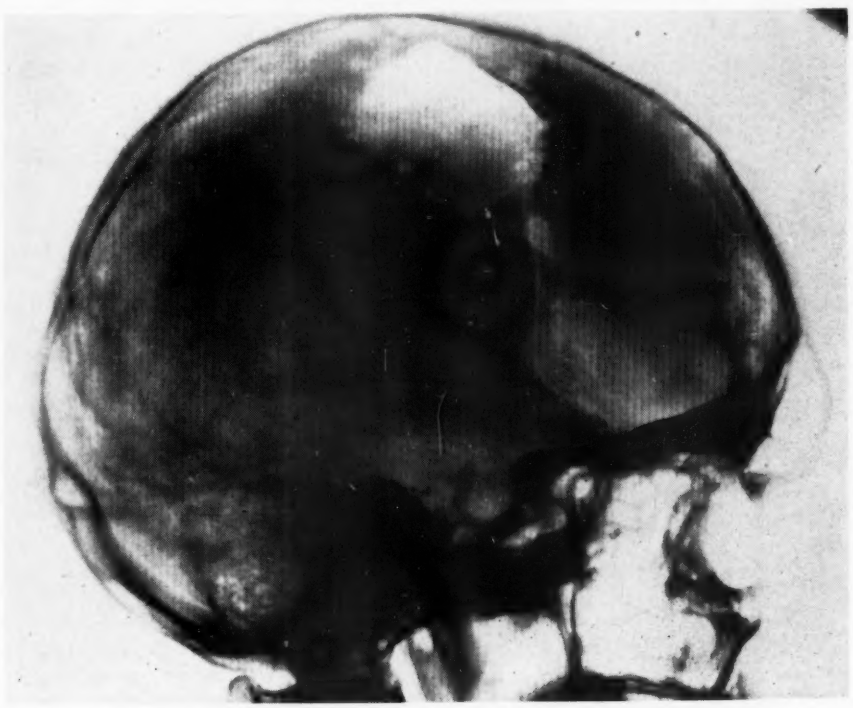

Fig. 17 (Case 17).—Lateral roentgenogram taken nearly five years after the insertion of an ilium graft (February, 1945), showing that there has been little absorption and that bony union has occurred.

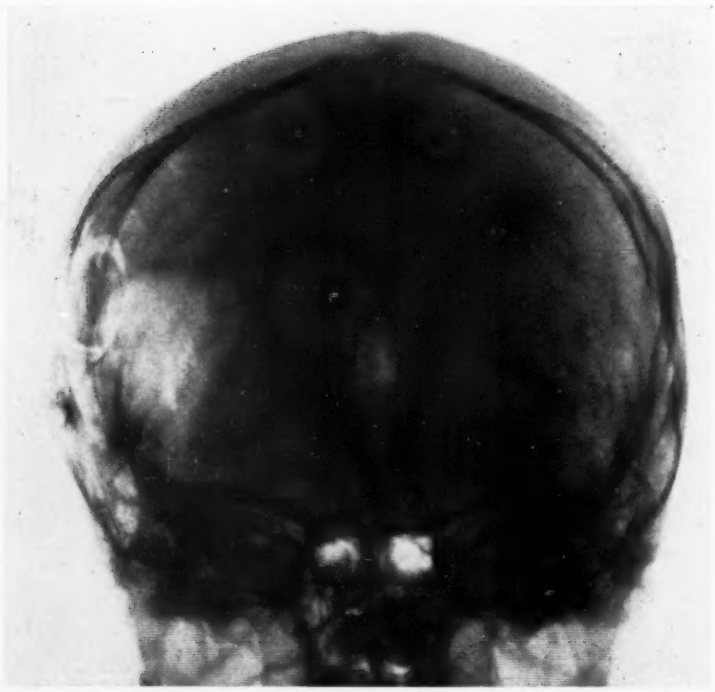

Fig. 18 (Case 19).—Anteroposterior encephalogram showing the extent of the diverticulum from the right lateral ventricle, almost reaching the site of the fracture.

Fig. 19 (Case 19).—Posteroanterior encephalogram taken with the patient sitting up, to show the upper and outer limits of the diverticulum.

bone formation, and evacuation of a collection of fluid deep to the inner layer of celluloid, relieved the severe headaches, but sufficient time has not yet elapsed for adequate follow-up.

The encephalograms accompanying Case 19 (Figs. 18 and 19) show the extreme degree to which a traction diverticulum from a ventricle may extend. In this patient, the scarred cortex was only a few millimeters thick, but it was possible to dissect it off the ependyma of the diverticulum and leave the latter intact. The insertion of a graft from the patient's ilium (Fig. 20) has relieved the major fits. Three petit mal attacks with visual hallucinations occurred when sedation was stopped too soon. He has, however, been able to continue his work in mechanical engineering.

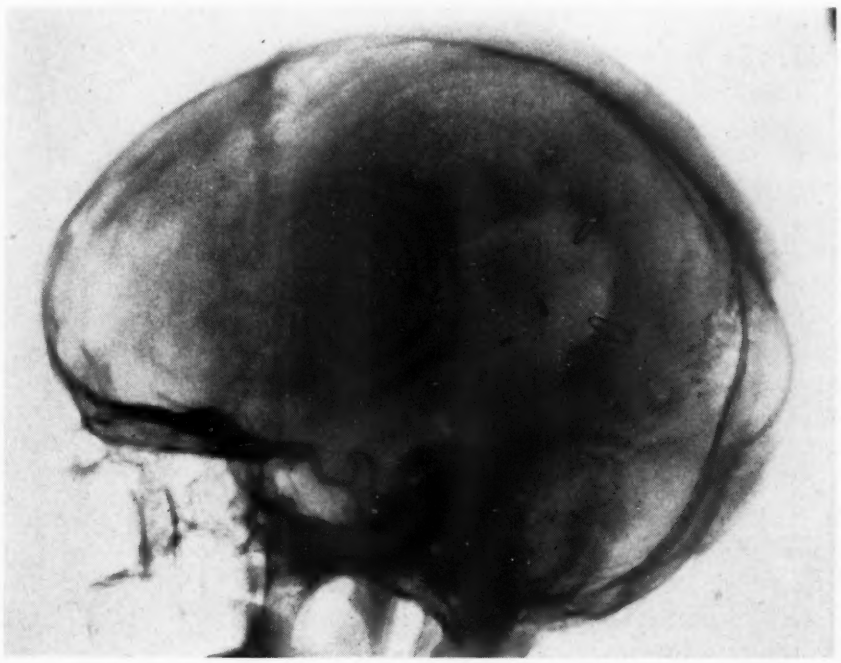

Fig. 20 (Case 19).—Lateral roentgenogram taken in February, 1945, showing the ilium graft wired into position nine months previously. There has been no absorption and bony union is seen to be occurring.

Complications were few. In three patients, infection occurred in the wound of the ilium and did not settle down until several large pieces of Horsley's wax had been evacuated. More careful preparation and storage of the wax, and a less liberal application to the diploic surface of the ilium eliminated this trouble.

In one patient (Case 2) in whom part of the large graft contained the whole thickness of the ilium, during a nocturnal fit one month after operation, a fracture of the ilium was caused by muscular violence. Outward and downward displacement of the fragment was produced by the action of the gluteal muscles. It was replaced by manipulation and kept in place with a plaster cast. After six weeks firm union and a satisfactory result occurred.

The only death in the series was due to a streptococcal infection of a hematoma under the scalp flap. Subsequently, a secondary infection with a

hemolytic *Staphylococcus aureus* also took place. Sulfanilamide and sulfapyridine were both administered early, but, in the light of more recent knowledge, the initial dosage was probably inadequate, and the organisms became sulfonamide resistant. In spite of these drugs and removal of the graft, the infection spread to the meninges and to the ependyma of the ventricle whose diverticulum had been opened into widely, with a fatal result about four weeks after operation. The graft itself, though from a cadaver bone, was not incriminated.

Unusual complications occurred in Case 7, not directly concerned with the bone grafting. About one year after the second operation, and associated with an attack of furunculosis, the patient was found to be suffering from diabetes mellitus which could be controlled only with large doses of insulin. As improvement occurred, several hypoglycemic episodes followed, during one of which he had a violent fit and fell downstairs. Some weeks later, a kyphosis was noted in the thoracic region, and compression fractures of the bodies of fourth, fifth, and sixth thoracic vertebrae were discovered in a roentgenogram of the spine. In July, 1942, he died of diabetes mellitus.

RESULTS

In eleven patients (55 per cent) "satisfactory" results have been obtained in that, for periods from nine months to thirteen years after operation, they have been symptom free and at continuous work. Of these patients, six had had epileptiform fits. Thus, 46 per cent of the patients suffering from traumatic epilepsy appear to have been cured.

Five patients, of whom four had had fits, were classified as "relieved," in that their symptoms and fits occurred with less frequency and severity. In two patients (Cases 7 and 15) no relief of fits occurred after depressed fragments of bone, foreign bodies, and adhesions were merely dealt with; the brain scars were excised later. Relief was then afforded the former. Unfortunately, the latter died.

Two patients had been classified as "unsatisfactory." In one (Case 10) the fits recurred three years after insertion of a cadaver graft, but if the second operation with ilium graft is more successful, he may be upgraded later. In another (Case 12), after one year's freedom, fits recurred upon cessation of sedation, but he was able to serve three years in the Armed Forces. The graft in this case was also composed of cadaver bone, and a second operation to insert a graft from the ilium bone is contemplated. Finally, in one patient who had not had fits, follow-up was only for a few months and has not been classified.

FATE OF THE GRAFT

Where cadaver bone has been used, varying degrees of absorption of the graft have taken place. In Case 12, the roentgenogram (Fig. 15) taken nearly six years after operation does not show any radiologic evidence of the graft, which appears to have been completely absorbed. The defect, however, felt quite solid and was not tender. In Case 10, absorption was extensive, but not quite complete in six years (Figs. 12 and 13). There was much less absorption in Case

14, as can be seen from the roentgenogram (Fig. 16) taken five years afterward; in Case 8 only a few small areas of absorption were noted after nearly five years (Figs. 7 and 8).

The fate of the ilium grafts has been more satisfactory. In Case 17, the roentgenogram (Fig. 17) taken nearly five years after operation shows that the graft is practically intact and that bony union has occurred at many places around the periphery. In Case 2, although some absorption has taken place during the thirteen years which elapsed (Fig. 2), the clinical and cosmetic result has been satisfactory. In Case 19, after a period of nine months, the graft looks intact and bony union has already occurred (Fig. 20). Other patients have been traced by letter, and their clinical results are uniformly good. Furthermore, in Case 7, where a second operation was performed twenty months after the graft, for excision of the brain scar, the graft was found intact, and bony union had occurred between it and the skull, except at two small areas. Thus, the chances of survival of ilium grafts appear to be excellent.

Similarly, where the outer table of a large fragment of the skull was used (in Case 9), the graft has undergone very little change (if any) after six years (Figs. 9 and 10), so that this form of grafting also appears to be satisfactory, when available.

SUMMARY

1. The sequelae of cranial defects, both organic and psychologic, are discussed, and reasons for their repair are given.

2. Some historic aspects of the various methods of grafting are referred to, and attention drawn to the different sources of grafts (a) from bones, both autogenous and homogenous, and (b) from inorganic materials, such as celluloid, metals, and plastics.

3. The advantages of using the inner table of the ilium bone over other bones and various inorganic substances are pointed out.

4. Indications for bone grafting to the skull are given and important points in the operative technique and its scope, are described.

5. Table I is submitted, giving details of a series of 20 consecutive patients treated with 21 grafts. In 16 patients, laceration of the dura mater and/or brain had occurred; in 10 patients, brain scar and ventricular diverticulum had developed; 13 patients had suffered major epileptiform fits. Special comment is made upon the unusual features of some of the cases.

6. The inner table of the patient's ilium was used on 14 occasions, and the outer table of the skull, 2 times. Cadaver grafts were used in the remainder. On 3 occasions, a free graft of fascia was used to repair or replace damaged dura mater.

7. The follow-up results for periods from thirteen years to nine months are presented: 11 cases have been classified as satisfactory; 5 as relieved, 3 as unsatisfactory'' (including one death), and 1 was not traced long enough to be classified.

8. Of the 13 patients who had fits, 6 had been regarded as cured, and 4 as relieved.

The assistance of the various resident medical officers and anesthetists who performed their duties so ably in these long and often tedious operations during the period under review is gratefully acknowledged.

The Australian Broadcasting Commission and the Sydney Press helped materially in tracing some of the patients. Mr. Woodward Smith of the Department of Medical Artistry, University of Sydney, took the portraits used in Case 5, and Mr. J. J. Fallon of Sydney kindly made the prints from the various roentgenograms reproduced.

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CEPHALHEMATOMA DEFORMANS

Late Developments of Infantile Cephalhematoma

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HEAD injuries acquired before, during, and just after birth, in infancy and in childhood, are now generally recognized as important causes of severe cranial and cerebral lesions leading to mental abnormalities, epilepsy, mono-, hemi- or diplegia, blindness, or deafness.

The sequelae of infantile head injuries are somewhat different from cranial and intracranial post-traumatic lesions in adults; they are as follows:

1. Fractures of the infantile skull may not heal by bony union; on the contrary, the dehiscence between the margins of the fracture may enlarge, thus producing a considerable cranial defect after some years. Probably this is due to general or local increase of the intracranial pressure (*meningocele spuria traumatica Billroth; hernia cerebri traumatica*).

2. Subpericranial and extradural collections of blood (*cephalhaematoma neonatorum* and *infantum*) sometimes produce in their late stages cystic cavities, osseous shells, or tumorlike hyperostosis of the cranial walls.

3. An extensive ossification occasionally takes place inside organized subdural hematomas following infantile head injuries.

4. Cerebral or meningeal hemorrhages of early childhood often cause diffuse or localized shrinkage of the brain (traumatic porencephaly and microgyria) with consequent enlargement of the ventricles or formation of cysts containing cerebrospinal fluid.

5. Uni- or bilateral micro- or hydrocephaly, generalized or localized thickening or thinning of the cranial walls may develop in childhood due to post-traumatic scar or cyst formation of the intracranial contents.

6. It is not infrequent that characteristic calcifications called "brainstones" are formed during childhood in traumatic scars of the brain.

Roentgenologic examinations afford the best opportunity to study the development of the post-traumatic cranial lesions mentioned. The roentgen findings are often significant in cases where the history and clinical examinations are not very helpful for diagnosis. Dandy's encephalographic method is of outstanding importance for the detection of intracranial abnormalities due to infantile head injuries.

In this paper we are dealing with the clinical state and the roentgen appearance of the late developments of infantile cephalhematoma. It is well known that infantile cephalhematoma persists for weeks or months and then, as a rule, completely disappears. Occasionally in the later stages hyperostosis or cyst formation of the cranial walls takes place; to this peculiar organization of cephalhematoma Recklinghausen gave the name osteitis fibrosa cystica. Choyce[1] stated: "An ossified haematoma might simulate leontiasis or tumour of

the skull, but will be distinguishable in a skiagram and by palpation, if, as likely, ossification is incomplete over the summit of the swelling." Chorobski and Davis[2] gave a detailed review of clinical and surgical experiences regarding cephalhematomas. Schüller[3] described x-ray findings in a case of cephalhematoma frontale.

In the case reports are presented clinical histories and x-ray findings in five cases with diagnoses of late development of infantile cephalhematoma.

Fig. 1 (Case 1).—Lateral roentgenogram of the right side of the skull showing hyperostosis of right frontal area.

CASE REPORTS

CASE 1.—Miss M., aged 27 years, complained of occasional headaches for three years. When 9 years old she was hit on the head by a falling blackboard; a swelling which has persisted since developed quickly on the right side of the forehead. On examination the right side of the forehead, mainly in the supraorbital region, and the adjacent temporal area were found to be prominent; the right eyeball stood lower than the left. The scalp could easily be moved over the smooth, nontender prominence. There were no objective or subjective murmurs. Smell and eyesight were normal.

Radiograph of the skull (Fig. 1) showed thickening of the right side of the forehead up to 16 cm., corresponding to the major part of the vertical plate of the frontal bone. No sharp outline could be seen between the thickened and neighboring normal part of the cranium. The diploe of the thickened area appeared partly normal, partly rarefied, and contained scattered dense islands 2 to 5 mm. in diameter. The internal lamina was thin and dense, the external lamina well outlined but thinned or absent over the summit of the bulge.

The frontal sinus was absent on the right side; the left frontal sinus was normally developed as were the other sinuses on both sides. The horizontal plate of the frontal bone on the right side was thickened up to 12 mm. and had a dense structure; it formed a flattened roof over the right orbit, the level of which was 1 cm. below the level of the left orbital roof.

CASE 2.*—Mrs. B., aged 54 years, was sent to the Melbourne Radiological Clinic for x-ray examination of the chest and paranasal sinuses. An abnormal appearance of the frontal bone was detected. Clinical examination showed a tumorlike prominence in the right frontoparietal region. The swelling followed a fall on the floor when the patient was some weeks old; it had never caused trouble. X-ray examination of the cranium (Figs. 2 and 3) revealed the following details: The medial part of the right parietal bone and the vertical

Fig. 2 (Case 2).—Lateral roentgenogram of the right side of the skull showing hyperostosis of right frontal area.

portion of the right frontal bone were thickened. The appearance of the internal and external laminae of the thickened area was not much different from the normal side. The diploe had a mottled appearance due to the presence of osteoporotic and sclerotic islands. No sharp outline could be seen between the mottled area and the normal cranium. The frontal sinus was absent on the right side; it was of medium size and normal shape on the left side. There was also a pronounced difference between the two halves of the anterior part of the base. The horizontal portion of the frontal bone, the posterior part of the ethmoid, the body, and the lesser wing of the sphenoid were much thicker on the right than on the left side. The roof of the orbit stood on a lower level than on the normal side. The thickened right lesser wing of the sphenoid was of uniform dense structure; the thickened right half of the sphenoidal body and the right posterior ethmoid were partly dense, partly pneumatized. A supraorbital extension of the right ethmoidal cells filled the anterior half of the right horizontal plate of the frontal bone.

*History and radiographs of this patient through the courtesy of Dr. Stuart Cross.

In Cases 1 and 2 there was similar roentgen appearance, namely, (1) thickening of the right anterior part of the calvaria presenting a mottled diploetic structure, (2) eburnated hyperostosis of the right anterior part of the base of the skull, (3) an atypical paranasal pneumatization on the right side.

Fig. 3 (Case 2).—Posteroanterior view showing hyperostosis of right side.

CASE 3.—Mrs. O'D., aged 72 years, was sent to the x-ray department of St. Vincent's Hospital for examination of a cranial deformity. The patient herself did not want this examination as she had never been inconvenienced by the cranial deformity. In her early childhood she had been hit on the head by an axe; a cranial deformity developed after the injury. The photograph of the patient (Fig. 4) shows an asymmetrical prominence of the forehead and the naso-orbital region; both eyeballs appear displaced laterally, the left one more so than right. Enlarged veins could be seen on the skin covering the left frontotemporal prominence. The left eyebrow was elevated. On palpation the bulge was resistant like bone and not tender. The skin could be easily folded over the bulge.

Radiograph (Fig. 5) showed an enormous thickening of the anterior part of the cranial wall; the tumorlike prominence was mainly eccentric and more pronounced on the left side. The largest diameter of the thickening was about 6 cm. Lamina externa and interna were well outlined and smooth. The space between the two laminae contained cystic cavities, normal and porotic diploe, and numerous islands of dense bone, some of which looked like eburnated sequestra. No frontal sinuses could be seen. In the base of the skull the pos-

Fig. 4 (Case 3).—Photograph showing deformity of face and skull.

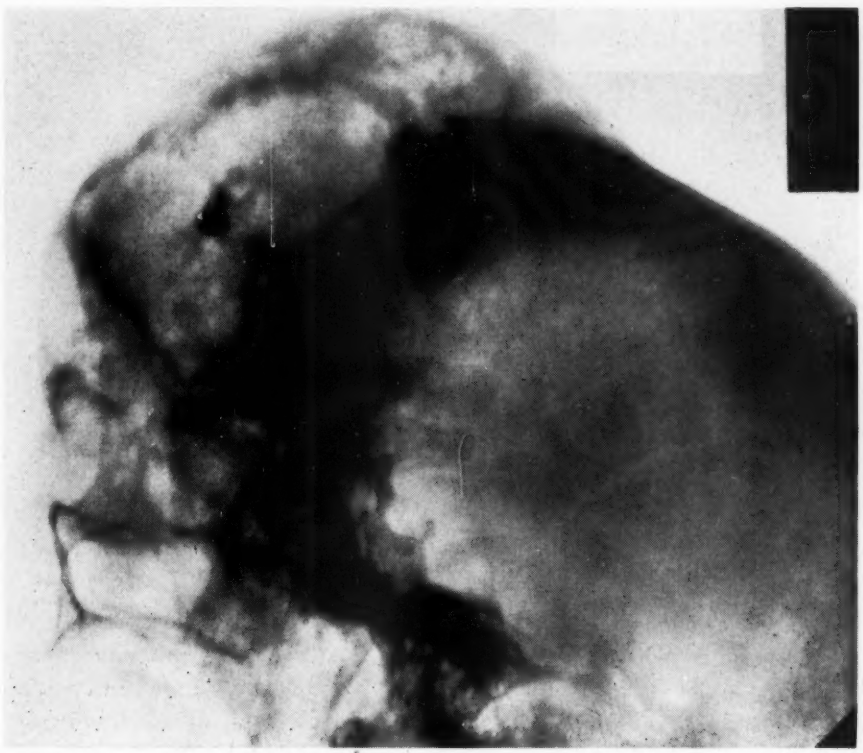

Fig. 5 (Case 3).—Lateral roentgenogram of the right side of the skull showing extreme deformity and hyperostosis.

terior part of the ethmoid and the anterior part of the sphenoidal body presented an enormous enlargement up to 6 cm. in the transverse diameter between the orbital cavities (hypertelorism). The structure of the enlarged parts was dense on the left side; pneumatization of the ethmoid and sphenoid was present on the right side. The great wing and the pterygoid process on the left side were thickened and dense. The horizontal parts of the frontal bone had a peculiar appearance: Profile radiographs showed the outlines of the cranial and orbital plates distinctly on the right side; the two plates diverged from behind forward at an angle of 45 degrees and were separated from each other by spongy bone; on the left side the horizontal portion of the frontal bone appeared thicker and denser than on the right side; its orbital plate was indistinctly outlined.

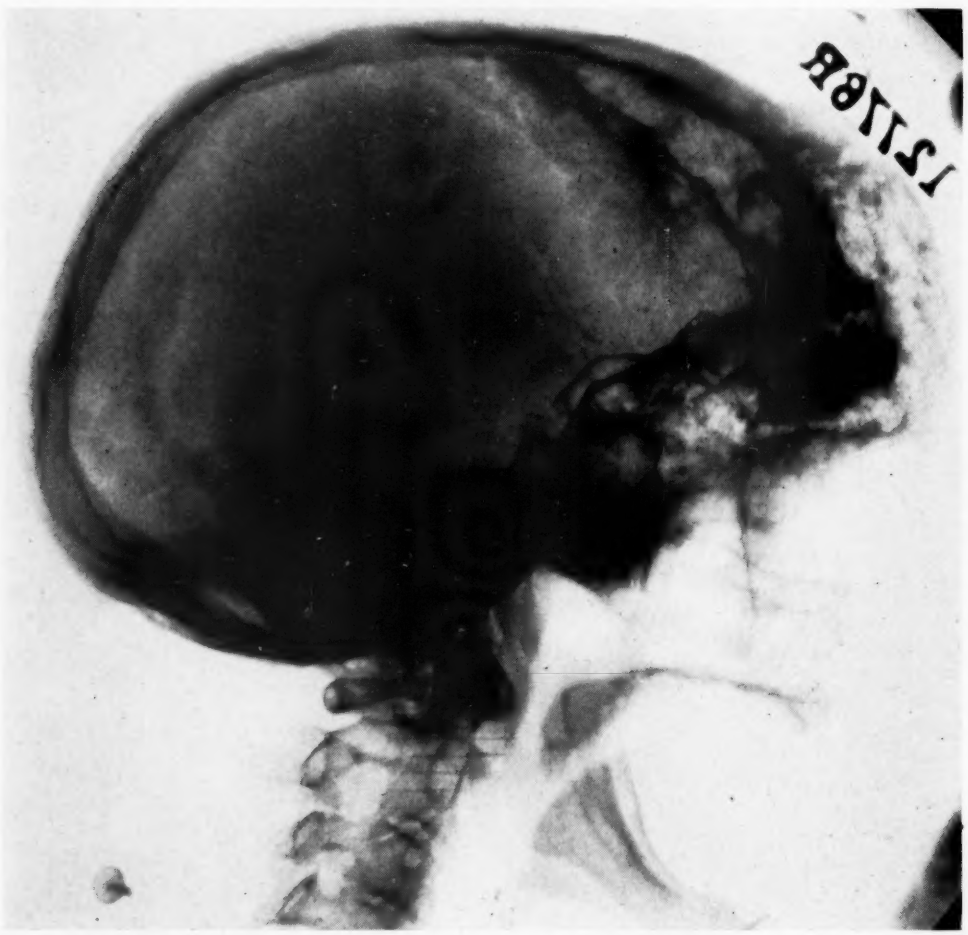

Fig. 6 (Case 4).—Lateral roentgenogram of the right side of the skull showing marked hyperostosis.

In Case 3 the extent of the asymmetrical hyperostosis occupying the frontotemporal region and the anterior basal part of the skull was enormous. The roentgen appearance of the cranial deformity was bizarre and offered some difficulty of explanation. Nevertheless, the peculiar type of the deformity and its structural abnormalities could be well interpreted by comparison with the findings in Cases 1 and 2, especially with regard to the mottled appearance of

the diploetic hyperostosis of the calvaria and the dense hyperostosis of the base. An excessive degree of hypertelorism was present in this case: the orbital cavities were 6 cm. apart. Pneumatization of the frontal, ethmoidal, and sphenoidal bones was absent on the left and poorly developed on the right half of the skull.

CASE 4.—Mr. R., aged 71 years, presented a pronounced deformity of the forehead, noticed since early childhood; it did not cause any trouble. X-ray examination of the head (Fig. 6) showed thickening of the anterior half of the cranium and of the base of the skull. The hyperostosis was more pronounced on the right side. No paranasal sinuses were present on the right side, but there were large ones on the left. The structure of the hyperostosis was similar to that of Case 3.

The comment given on Case 4, when published by one of us (A. S.)[3] in 1940, was as follows:

"With regard to the history of the case, especially the presence of the deformity since early childhood and the absence of clinical symptoms during the whole life, the most probable diagnosis is: Cephalhaematoma.

Fig. 7 (Case 5).—Photograph showing exophthalmos and deformity.

"We can imagine that in early childhood a large traumatic haematoma elevated the outer periosteum of the anterior half of the skull. Later on, the wall of the haematoma was ossified, forming a cystic cavitation. Inside the blood clot different types of osseous structure have developed, spongy bone, nodules and ridges, but especially a most unusual looking new growth-like hypertrophy of the walls of the rudimentary sinuses."

CASE 5.*—Miss P., aged 20 years, complained of double vision and prominence of the left eye seven weeks before admission to the Cancer Hospital, London. Three weeks later an aching pain developed in the left supraorbital and temporal region. This region was injured in childhood by a cricket ball. The injured area of the skull had since been prominent. During the four weeks previous to admission to the hospital the swelling had increased in size. After admission the pain disappeared and the swelling became smaller after rest in bed.

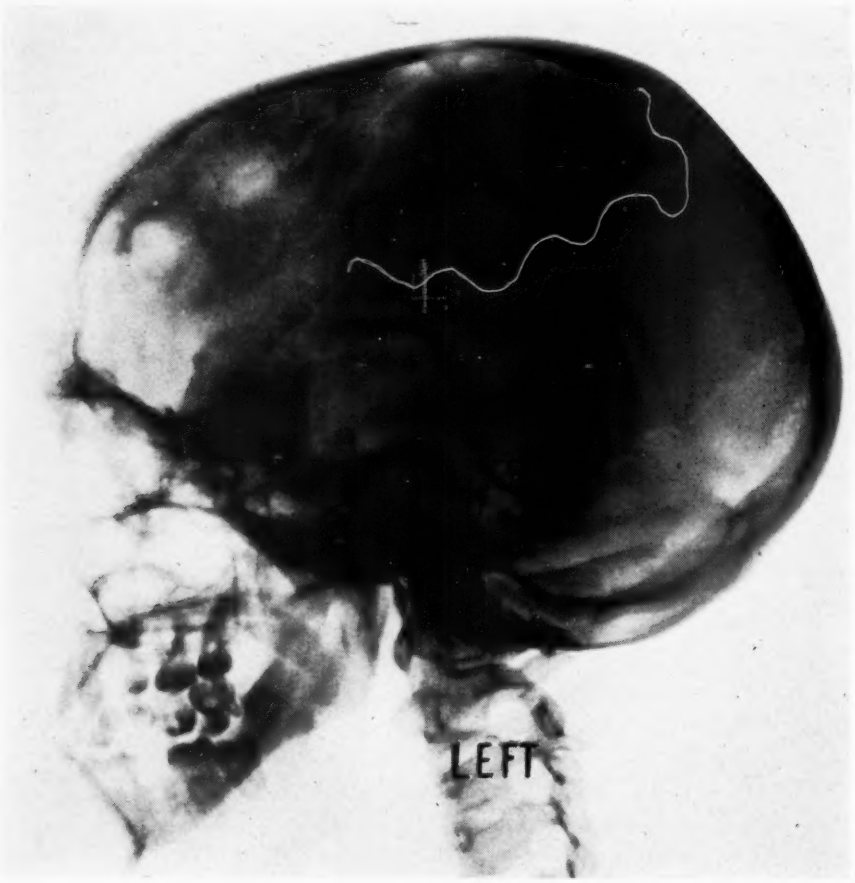

Fig. 8 (Case 5).—Lateral roentgenogram of the left side of the skull showing large defects and hyperostosis.

Examination showed a diffuse enlargement of the left frontal and temporal region (Fig. 7) and a small shallow depression about one inch in diameter above the left supraorbital margin. The skin over the whole region was normal, neither fixed nor tender. The bone felt soft like a fetal skull at the site of the shallow depression mentioned previously. The left eyeball was displaced forward and downward. The lacrimal gland felt hard and was pushed forward. The actual movements of the eye were normal apart from disability due to alteration in position. Vision was normal; there was no pulsation of the eyeball; no bruit was audible.

Blood and urine examinations were normal. Wassermann test was negative, inner organs were normal, and radiographs of the skeleton negative.

*Clinical history of this patient, photographs, and radiographs were sent to one of us (A. S.) by the late Professor I. M. Woodburn Morrison.

A small piece of bone was removed from the left frontal region at the periphery of the swelling. The trephine plug was examined by the pathologist. He reported that the associated features of the histology of the specimens and the failure to detect any trace of a neoplastic or granulomatous process suggested that the condition was primarily pressure necrosis of the bone, probably of traumatic origin, resulting in supracranial or intracortical hemorrhage which failed to organize and induced a rarefying osteitis in the neighboring bone; the vascularity of the loose connective tissue filling the spaces would be liable to increase the absorption of bone and to favor hemorrhages. Massive hemorrhage into coalescing residual cysts following such hematomata would tend to occur.

Radiographic examination of the skull (Fig. 8) showed the following details: The bulge of the left forehead was caused by thickening of the vertical portion of the frontal bone. A large defect of the bone was present corresponding to the depression in the left supraorbital region. The defect measured two and one-half inches in the vertical and two inches in the transverse diameters; the outline of the defect was irregular. The structure of the surrounding thickened part of the frontal squama appeared diploetic and mottled by spotted sclerosis. Frontal sinuses were wanting. On the base of the skull there were marked changes in the walls of the left anterior and middle fossae. The roof of the left orbit was absent. Its defect appeared to be continuous with the large defect in the vertical portion of the frontal bone. The greater and lesser wings of the sphenoid on the left side were thickened and of dense structure. The left half of the sphenoidal body was not pneumatized; the sphenoidal sinus on the right side was well developed.

In Case 5 are shown the characters of unilateral frontal hyperostosis similar to those in Cases 1 and 2. Case 5 varies from the other two in that there was a large defect in the central portion of the hyperostosis. Parts of the vertical as well as the horizontal plate of the frontal bone were destroyed. The destruction was probably caused by a recent hemorrhage inside the hyperostosis.

DISCUSSION

The histories, the clinical signs, and the radiographs of five adult patients have been described, showing similar and characteristic features:

1. A history with origin in infancy or childhood (a cranial injury is mentioned as the cause in four of the five cases)
2. A peculiar type of bulging of the forehead, more pronounced on one side
3. A circumscribed cranial hyperostosis presenting an unusual and bizarre structure

No sign or symptom of intracranial or ocular disorder was present in Cases 1 to 4. The deformities of the head which endured from youth to adult life did not worry the patients. The ages of the four patients when examined were 72, 71, 54, and 27 years.

One patient (Case 5) entered the hospital because of headaches and protrusion of the eyeball suddenly at the age of 20 years. A defect of the frontal bone in the center of the hyperostotic area was detected by clinical and radiographic examinations. This defect was probably due to fresh hemorrhage. The sudden onset of symptoms can be attributed to pressure by the hematoma. This explanation is supported by the result of the biopsy.

One patient (Case 4) was a man, the others were women.

The roentgen appearance of the peculiar type of circumscribed hyperostosis can be summed up as follows: The calvaria and the base of the anterior part of the skull are involved, mainly on one side. These parts are thickened considerably, up to 6 cm. in one case. The thickening is mainly eccentric, its ill-

defined edges merge gradually into the normal neighboring bone. The structure of the hyperostosis is not uniform; diploe lined by thin external and internal laminae prevails in the thickened calvaria, eburnated hyperostosis in the basal area. One may find osteoporosis, osteosclerosis (similar to the well-known structure of the calvaria in Paget's osteitis deformans), cystic cavities, and bony fragments resembling sequestra inside the bony swelling of the calvaria. Sometimes paranasal sinuses are not developed on the diseased side; if they are, their shape is atypical.

None of these patients required operative treatment.

We propose for the cranial deformities represented by the five cases described here the name "Cephalhematoma deformans."

The pathogenesis of cephalhematoma deformans can be explained as follows: Early stages of infantile cephalhematoma occasionally show new formation of bone along the elevated pericranium, along the displaced dura mater, and inside the swelling. The newly formed bone may completely disappear; rarely it persists and grows *pari passu* with the growth of the normal part of the skull. Osteoporosis or osteosclerosis and formation of sequestra or cysts may develop inside the swelling. Paranasal pneumatization is absent or atypical. Fresh hemorrhage may occur and partially destroy the swelling.

Differential diagnosis must eliminate various types of cranial thickening: osteoma, chondroma, sarcoma, leontiasis ossea, meningiomahyperostosis, hyperostosis combined with facial nevi and angiomatosis, syphilitic hyperostosis, hyperostotic type of xanthomatosis, hyperostosis in neurofibromatosis Recklinghausen, osteitis fibrosa cystica Recklinghausen, Albright's disease, hyperostotic oxycephaly, hypertelorismus Greig, mucocele of the paranasal sinuses, cholesteatoma of the cranial bones, cholesteatoma of the paranasal sinuses.

SUMMARY

This paper deals with the late developments of infantile cephalhematoma.

Five patients, one man and the four women, at the ages of 20, 27, 54, 71, and 72 years, respectively, showed a peculiar type of cranial deformity—a unilateral bulging of the anterior part of the skull.

It was unusual for the patients to complain of symptoms referable to the cranial abnormality.

X-ray examination revealed an extensive hyperostosis as the basis of the deformity. The anterior part of the skull was involved. The structure of the hyperostosis was not uniform; diploetic hyperostosis prevailed in the calvaria, eburnated hyperostosis in the basal area; osteoporosis and sclerotic islands, large sequestrum-like fragments of bone and sharply outlined cavities inside the diploe were characteristic features of the structure. Pneumatization was wanting or atypical in the hyperostotic area.

The name cephalhematoma deformans seems adequate for this peculiar type of hyperostosis.

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CONCUSSION AND CONTUSION

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THE only way in which mechanical forces have been shown to cause a reversible block of nerve conduction is by straining nerve fibers to a critical extent. Romanes recorded a condition similar to concussion in medusae which lay on an anvil while it was repeatedly struck. This does not happen with slugs and snails even when immersed in a water bath with the anvil as the floor. Even with suprasonic vibrations which one would expect to be the ideal system of producing commotion in cells, the effect appears to result from bubbling, producing local stretching, or from heating.

With this fact that strain is the only mechanical method of producing reversible nerve block as a basis, we can now consider the present knowledge of the mechanism of production of reversible and irreversible injury of neuraxial tissue by mechanical forces. Denny-Brown and Russell showed that if the head is allowed to move freely under the impressed force the threshold for injury of a given degree is lower than when this is resisted by a counterforce, that is, the energy application for concussion is less when the applied force produces free acceleration of the head than when it produces deformation of the head only.

The following are possible results of applying force to the free stationary head:

1. A linear acceleration of the whole skull and its contents in the line of the applied force.

2. An angular acceleration of the whole skull and its contents about an axis within or without the cranial boundaries.

3. Distortion of the skull with production in the rigid resilient bony structure of longitudinal and transverse waves which are highly damped by the liquid loading of the contents and surroundings.

4. Distortion of the skull with production of general change in pressure within it. With an ellipsoid of revolution bounded by a rigid resilient wall, reduction of the long axis results in a generalized decrease of pressure in the content, and reduction of a short axis results in a generalized increase in pressure.*

The skull may not, however, behave simply as an ovoid bottle. The curious sliding surface between the squamous temporal and neighboring bones suggests considerably more complex possibilities. There is not at present any mechanism which could be relied upon to register intracranial pressure on impact. That used by Denny-Brown and Russell is completely inadequate.

In most cases of application of force to the skull, the result will be a combination of all these possible changes. The proportion of the applied

Aided by a grant from the National Health and Medical Research Council.

*This feature of ovoid bodies was pointed out to me by Professor W. A. Osborne.

energy appearing in each change will depend on the velocity (vector), mass, and physical properties of the impinging object and on the physical characters and the spatial relations of the part of the particular skull to which it is applied.

The anatomic attachments of the skull are of the greatest importance. It is always attached to the neck which can be bent but has an axial constituent of great strength, the vertebral column. This is broken up into segments of rigid bone joined by strong but elastic structures, the ligaments; attached to it are masses of viscous liquid material. Thus, a force applied along a line which would produce linear acceleration in an unattached skull may produce an angular acceleration as well as a rectilinear acceleration of the attached skull and the linear acceleration will always bear some relationship to the axis of the neck.

Omitting for the moment the physical changes produced within the skull during acceleration and treating it as moving as a whole it is important that although the skull is joined to the vertebral column by strong but elastic ligaments, the intracranial neuraxis is joined to the spinal cord by the weak spinomedullary junction with an extremely low coefficient of restitution. Denny-Brown and Russell stated that in experimental cats they produced an acceleration from a velocity of 0 up to 28 feet per second in $\frac{1}{1,000}$ of a second; this is approximately 1,000 g and very improbable. Accepting it as a basis for simple calculations, however, we have also the average weight of the spinal cord of six cats as 7 Gm. If the skull and its contents in their experiments moved directly away from the spinal column the force at the spinomedullary junction due to the inertia of the cord would be approximately 6.2 kg. weight. A very small fraction of this would be sufficient to produce functional impairment of this junction. This analysis can be applied to acceleration not in direct line with the spinal cord by working out the components.

The threshold which Denny-Brown and Russell have stated for the cat is an acceleration from standstill to 28 feet per second in $\frac{1}{1,000}$ of a second. Actually, neither of these figures was the result of measurement. The final velocity of the head was probably in the neighborhood of 40 per cent of that reported by the authors (see Appendix), that is, the critical final velocity for cats was 11.2 feet per second, not 28 feet per second. The time in which this was produced cannot even be guessed at. To check on the figures, cats were anesthetized by inhalation of ether and suspended from a crossbar by copper wires entered under the planum occipitale at the external protuberance and emerging at the temple after passing deep to the zygomatic process of the temporal bone. The crossbar was allowed to fall on a taut guide wire to a length of piping screwed to a floor joist (Fig. 1). The time of deceleration was not measured but was almost certainly greater than $\frac{1}{1,000}$ of a second. A fall from 4 feet was sufficient to cause jerking, disorder of respiration, and loss of corneal reflex in five cats. A fall from 5 feet caused death with bleeding at the spinomedullary junction in all but one of six cats. The critical velocity in these cases was therefore about 16 feet per second.

To check whether the distortion at the atlanto-occipital region is the origin of the nervous lesions in these cases, cats were similarly suspended but completely immersed in a can of water. A tracheal cannula and rubber tube gave a respiratory pathway through the side of the can, and the buttocks of the cat rested on the bottom of the can. On dropping this can from a height of as much as 12 feet (terminal velocity, 27 feet per second), no signs of concussion were detected. In this case the time for deceleration was measured with a high-speed moving picture camera (2,300 frames per second) and time

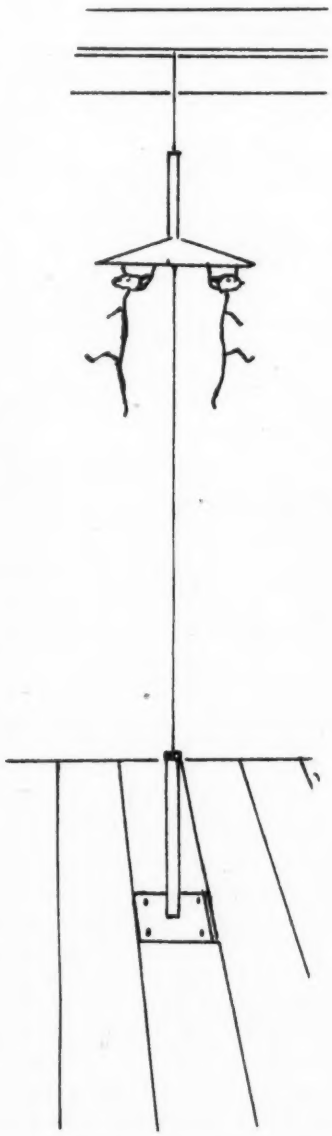

Fig. 1.—General setup for decerebration experiments. The cats are suspended by the skull to the crossbar which falls freely on the taut vertical wire until checked by the vertical stop screwed to the floor.

marker; it was $\frac{1}{1,600}$ of a second. This deceleration was half as great again as that claimed by Denny-Brown and Russell and probably six times as great as that described in the preceding experiments. In the case reported here, distortion at the atlanto-occipital region was reduced to a minimum and no disturbance of the nervous phenomena resulted.

As to whether such local distortion could result in unconsciousness we have the statement of Foerster[4] that local pressure (that is, distortion) on the neuraxis at this junction produces loss of consciousness more easily than does pressure applied anywhere else. It is thus obvious that acceleration and deceleration cannot be used interchangeably without taking the whole of the conditions into account. Thus, a cat with the head accelerated thus

is subject to the same forces as a cat decelerated thus

but not the same as a cat decelerated in this way:

In this latter system there is bony compression of the cervical vertebrae and the cord tends to invade a fixed volume, subjecting the spinomedullary junction to very little strain. In the former cases the ligaments are stretched and the spinal cord pulls on the spinomedullary junction.

Holbourn has advanced very good reasons for considering that angular acceleration may be a very important factor in causing intracranial damage. Briefly, the argument is that the moment of inertia of a particle of mass "m" at a distance "r" from the axis of rotation equals $mr.^2$ Thus, if we take the skull (Fig. 2) with an angular acceleration about an axis "a" within the skull, the resistance to acceleration of the two equal particles "m^1" and "m^2" is as $\frac{(am^1)^2}{(am^2)^2}$. It is obvious, too, that if the axis of rotation is outside the limit of the skull, for example at "a^1," the moment of inertia of "m^3" will be greater than that of "m^4," and therefore a movement of the liquid within the skull will result. The same principle applies as to the movement of fluids in semicircular canals. But the acceleration is being impressed on the viscous liquid contents of the skull by the skull because of its greater rigidity. Consequently, the part of the viscous content nearest to the inner layer of the skull, even though it may have the highest moment of inertia, will accelerate most nearly at the rate of the skull and the succeeding layers will tend to slide on each other. Where there are projections such as the alae pterygoideae there will be maximum distortion and macroscopic damage. But apparently Holbourn was not aware that Ferrari[4] had found that glass-walled chambers when disposed within the cranial content obliquely or perpendicular to the line of acceleration of the head were broken only when within 5 mm. of the inner surface of the skull. Although Ferrari did not analyze this in terms of angular acceleration it would appear to support the interpretation that as a result of angular

acceleration there is a strong shearing of the external layers of the cerebrum on the deeper layers. It is very probable that such a result over a large area of the cerebrum would result in unconsciousness in the milder trauma, and macroscopic damage in the greater trauma.

The third and fourth results of force applied to the head also need consideration. It is very easy to demonstrate with a piezoelectric crystal applied to the external surface of the intact cranium that transverse waves travel round the bony calvarium when the skull is hit (Fig. 3). It is probable that the energy imparted in glancing blows by small masses is dissipated mainly in this form. With sections of the skull as the walls of a ripple tank some idea of the pattern of internal pressure can be gained (Fig. 4). As the pressure waves in the content of the intact skull would all travel at the same speed it is probable that these ripple tank pictures approximate to the real distribution of foci of pressure change in the intact cranium.

Fig. 2.

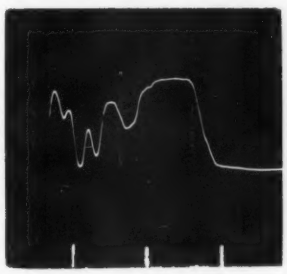

Fig. 3.

Fig. 2.—Diagram of skull showing effects of angular acceleration on intracranial contents.
Fig. 3.—Oscillograph trace from piezoelectric crystal on the temporal part of a human skull struck on the occiput, time marker: 1/100 sec.

The original premise was that the sole manner of injury of nervous tissue is by shearing of tissue. As Holbourn stated, the intracranial contents, when subjected to increased pressure, are as incompressible as water, but they do not show the same reactions to reduced pressures as does water. The blood contains 20 c.c. of O_2 and approximately 60 c.c. of CO_2 per 100 c.c. of blood. This gas is rapidly released and forms bubbles readily at approximately ½ atmosphere pressure when of course its volume is doubled. If such a release of gas takes place at a zone of low pressure, shearing of nervous tissue can readily occur. Whether this liberation of gas does in fact occur was tested by placing a blood clot in one end of a glass bottle of elliptical section, the remainder being filled with gelatin which was allowed to set. The side hole was then filled with paraffin and plugged. The end of the bottle opposite to the

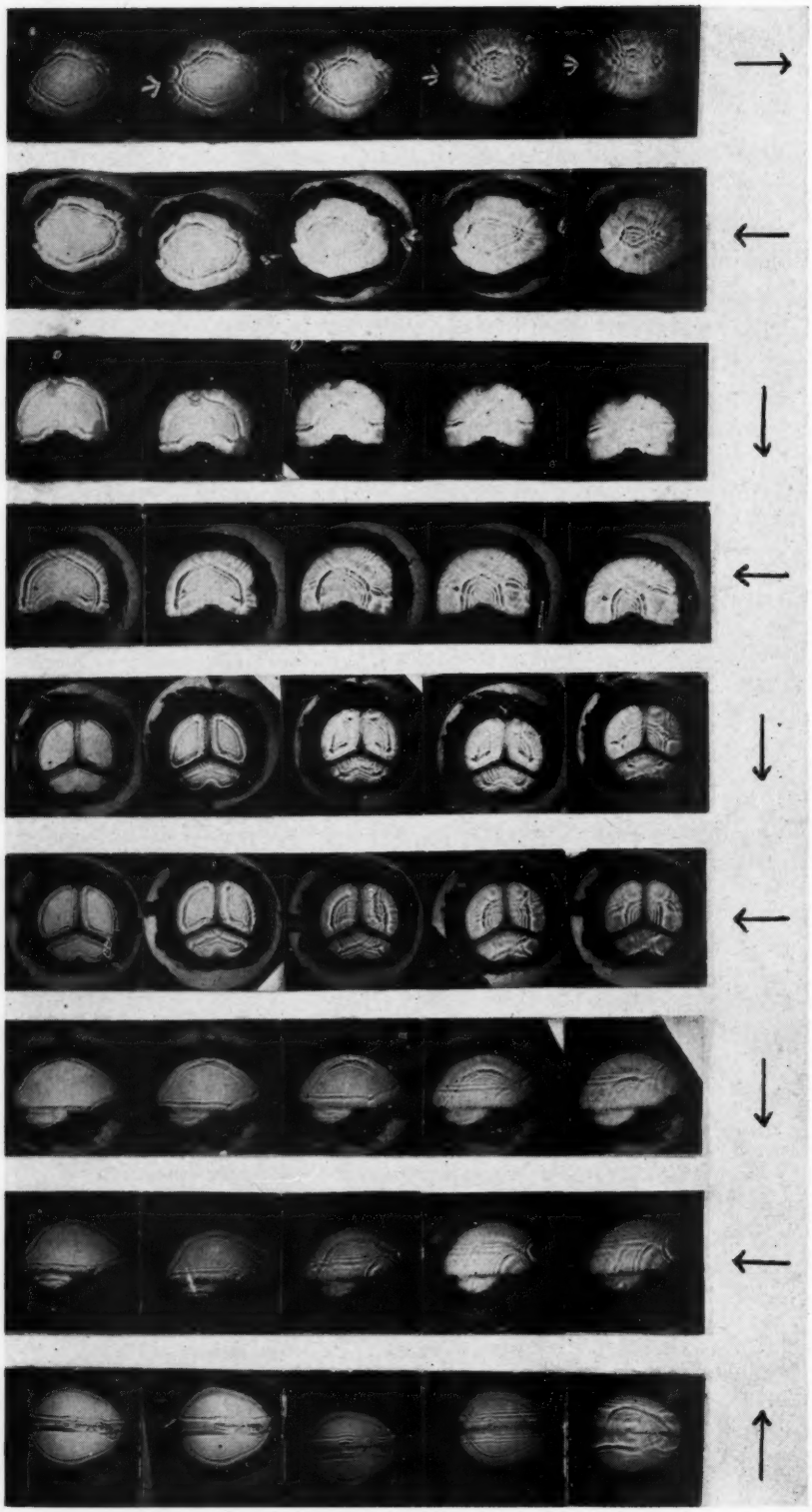

Fig. 4.—Ripple tank pictures of various sections of human skull struck in the directions indicated by the arrows. The ripple tank was made by cementing a section of skull 1 inch deep to a glass plate, and filling to ¾ inch with water. The photographs were taken by transmitted light and show the sequence of wave propagation. In series these are sections of horizontal, coronal of middle and posterior fossa, sagittal to side of midline and horizontal.

blood clot was then struck with a heavy rubber tire hammer. Fracture of the blood clot with residual bubbles was observed.

It is evident, therefore, that the three mechanisms mentioned can cause damage to the neuraxis. It would not be safe to conclude, however, that only by such damage does loss of consciousness result. Schmidt has shown that the loss of consciousness in dogs given sublethal doses of cyanide is due to action on the end organs of the carotid and aortic bodies. I have seen a fellow student rendered unconscious by pressure in the carotid sinus area for a duration of only a second or so. Breslauer records loss of consciousness from irritation of the gasserian ganglion in both man and dogs.

CONCLUSION

The cause of concussion and contusion is spatial distortion of the elements of the neuraxis. This may occur at the spinomedullary junction when the head is accelerated away from the neck. It may occur in the intracranial contents when the head is rotated (Holbourn). It may result from distortion of the cranium. It may also result from liberation of bubbles from the intracranial blood in areas of reduced pressure.

I wish to thank Dott & Co. for the blowing of special ovoid glass vessels, and Mr. Frank Nicholls for assistance in piezoelectric recording.

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(See next page for Appendix)

APPENDIX

CRITIQUE ON CALCULATIONS BY DENNY-BROWN AND RUSSELL*

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Assume striker and head of animal remain in contact after striking.

Let: h = distance of center of mass below axis of strike.
I = moment of inertia of striker about axis.
W_o = angular velocity of striker just before impact.
W = angular velocity of striker just after impact.
l = length of striker.
θ_o = initial inclination of striker.
m = mass of animal's head.
M' = mass of striker.
P = impulse of blow.
v = velocity acquired by animal.
v_o = velocity of strike.

$$\therefore M'gh\,(1 - \cos\theta_o) = \tfrac{1}{2} I W_o^2$$

$$P = mv$$

$$Pl = I\,(W_o - W)$$

$$v = lW$$

$$v_o = lW_o$$

Hence:
$$mvl^2 = I\,(v_o - v)$$

$$\frac{v}{v_o} = \frac{I}{I + ml^2} \qquad (1)$$

Hence, the velocity acquired by the head of the animal is less than the velocity of the pendulum on contact by a factor—

$$\frac{I}{I + ml^2}$$

For the pendulum of light steel tubing with 1.8 kg. weight 12 cm. from axle—

$$l = 100 \text{ cm.}$$

$$I = 1.8 \times 10^3 \times 12^2 + \frac{M}{3} \times 10^4 \text{ gm cm}^2$$

where M is the mass of the "light steel tubing."

Hence:
$$\frac{v}{v_o} = \frac{2.6 \times 10^5 + \frac{M}{3} \times 10^4}{2.6 \times 10^5 + \frac{M}{3} \times 10^4 + 10^4 m}$$

$$= \frac{26 + \frac{M}{3}}{26 + \frac{M}{3} + m} \qquad (1)_a$$

For M we have the information that the steel tubing was "light." Assume that it weighed 300 Gm. The heads of eight cats weighed 310, 230, 225, 220, 200, 170, 155, and 138 Gm. The mean weight was therefore 205 Gm. Substituting in $(1)_a$:

$$\frac{v}{v_o} = .38 \text{ for the mean value}$$

and ranges from .3 to .48. There is no statement in the paper that any allowance was made for this factor.

*Denny-Brown, D., and Russell, W. Ritchie: Experimental Cerebral Concussion, Brain 64: 93, 1941.

A HYPOTHALAMIC SYNDROME AND PERIVENTRICULAR EPILEPSY AS A LATE SEQUEL OF BRAIN INJURY

Report of a Case With Autopsy

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INTRODUCTION

ALTHOUGH many studies of the central nervous system have been made in those who have died after recent brain injuries, comparatively few have been made after many months or years have elapsed. The case reported here presented an opportunity for a detailed examination of such a brain.

The patient appeared to have died from a recurrent epilepsy of an unusual type, conforming in part to that described as periventricular or diencephalic epilepsy.[1, 2] These names connote an origin and type of discharge seemingly related to certain of the vegetative functions of the hypothalamus and brain stem. In this case there was ample evidence of disease in these zones at autopsy.

From the pathologic standpoint it is of value in showing what changes may be observed in the brain about fourteen months after a severe head injury of the closed type.

The case will be presented, with the pathologic findings, and thereafter the clinical and pathologic implications will be discussed.

CASE REPORT

S. McC., schoolboy aged 8 years, was referred to this hospital from Hobart, Tasmania.

Past History.—On June 30, 1943, when aged 7 years, he had been treated by another physician. When aged 11 months he had hemorrhaged after circumcision. At 13 months he had been operated on for hernia. He had always been considered delicate and had vomited easily. He walked at 13 months, but at the age of 3 years seemed weak in the legs. However, he had gone to school at 6½ years of age and progressed well.

Three months before the consultation he had been knocked down by a motor lorry. The site of the injury was not known, but he had been unconscious for three days, and semiconscious for four more. For three days he had screaming attacks in which the legs would stiffen and draw up to the chin. On the seventh day, when he became fully conscious, he was found to be paralyzed in the right arm and leg. For the first month he could not walk, although there was gradual improvement; however, he did not seem to progress over the next month. At first he had frequent headaches, which later disappeared, although an attack of headache and vomiting occurred eleven days before examination. He was considered by his parents as normal mentally. His speech had been affected for a time, but he had always been considered somewhat backward in this characteristic; it was now the same as before the accident. He walked fairly well usually, and his hand was unaffected. However, outdoors his walk was said to be far from normal, and he could scarcely run.

On examination he seemed well, although he walked with a slight limp. He did not cooperate well on neurologic examination, but it was noted that the right plantar response was extensor.

Illness at Time of Admission.—The patient was admitted to the hospital April 9, 1944. He had been attending school for six months, not missing a day until March. He had even attended until seven days before, but on the next day returned home since he did not feel well.

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About one month before, he had developed a voracious appetite, became very thirsty, and extremely sleepy. He had also gained much weight over this period, particularly about the neck. Since then he had suffered from attacks of "phlegm in the throat," which for the last week had troubled him greatly. At times, about one week before, breathing was abnormally rapid.

On March 8, 1944, at 9 A.M., he had a sudden attack in which his face became a high Turkey-red color with a touch of blue, although he was white about the mouth and nose. Hands and feet were blue and cold. A little later he perspired heavily, began to breathe quickly, and became sleepy or semiconscious. Gradually color returned, the entire attack lasting about one hour.

Afterward he was sleepy but could recognize his parents and often asked for food. At 12:30 he had a drink of water and asked for a biscuit, but after two bites he inhaled deeply and began to jerk his limbs, holding his breath for about one minute. At 1:30 P.M. he developed jerking attacks, with hands and feet clenched and eyes deviated, which started on the right hand and passed to the left. For about one-half hour he seemed semiconscious. He was rational at 3 P.M., but still asking for food and drink. At 4:30 he had a slight attack lasting for five minutes, but was semiconscious for ten minutes.

Phenobarbital, ¼ gr. hourly, seemed to relieve him, but all night breathing was very quick and pulse racing.

Next day he was brought from Tasmania to Melbourne by plane. At 7:30 A.M. he was quite conscious and breakfasted on porridge, tea, and toast. On the way to the plane by car he was sufficiently alert to recognize once that the driver was taking the wrong road.

After being in the plane for one-half hour he complained of being hot, and flung his arms about. Phenobarbital was given and he became drowsy.

Examination.—On May 8, 1944, examination at 6:30 P.M. disclosed an obese child, flushed and breathing rapidly. Respirations were 50 per minute, pulse rate 150, temperature raised. He seemed quite unconscious.

No abnormal neurologic signs were noted at this time other than a right extensor response. Earlier he had regained consciousness for a time, when the right knee jerk was said to have been weak, and a poorly sustained lateral nystagmus was present.

May 9, 1944, at 9 A.M., he was conscious, but the pulse was very rapid, and to 180 per minute. The respiratory rate had varied greatly, but at this time was from 7 to 12 a minute. The axillary temperature had varied from 103 to 104° F. His color was bluish, and respirations somewhat gasping. As his neck seemed swollen, adrenalin was given in case of glottic edema, but without effect.

The ventricles were punctured; the cerebrospinal fluid seemed under some increase in pressure and a cannula was left in the ventricle.

Death occurred on the same day.

*Autopsy.**—Heart, lungs, thyroid, neck, and abdominal and pelvic viscera were normal.

As the dura was incised the brain bulged through the incision. No gross abnormality was found on superficial examination, nor was anything abnormal found at the brain base, in the bones of the skull, or in the pituitary region.

Brain and pituitary gland were preserved in formalin for future examination.

Macroscopic Examination of the Brain.†—The surface of the brain after fixation showed no evidence of tumor, hemorrhage, or meningitis, and no cerebellar or uncal herniation. On sagittal section the following abnormalities were found:

(1) A large combined cavum septi pellucidi and cavum Vergae, its anterior extremity extending into the right frontal lobe, deformed the right anterior horn and burrowed well under the ependyma. Its lining was rough and granular. The right lateral wall was complete, but the left deficient in its posterior part.

*Done by Dr. H. Sissons and Dr. R. Willis.
†Examination by the author.

(2) Beneath the ependyma of the lateral ventricles were numerous small cavities, with ragged walls, some communicating with the ventricle. Elsewhere the ependyma over limited areas was separated from the underlying tissue, sometimes for several millimeters (Fig. 1).

(3) A collar of grayish tissue surrounded the sylvian aqueduct, and was particularly marked on its right side (Fig. 2). In places it was from 2 to 3 mm. wide. It could be traced to within 2 to 3 mm. of the quadrigeminal plate, and forward beneath the aqueduct into the hypothalamus, particularly on the right side. It did not seem to involve, or to extend beyond, the mamillary bodies. It could be followed caudally under the ependyma into the right side of the pons, where it became more superficial under the floor of the fourth ventricle at about its middle, and where it extended at least to the lateral angle of the ventricle. It could not be observed on the left side of the pons.

(4) A grayish, slightly spongy wedge-shaped area lay in the external capsule of the left hemisphere beneath the claustrum, with its apex toward the putamen, which may have been slightly involved. In coronal section it measured 1.8 by 4 cm. Its anteroposterior measurement was not made accurately, but was considerably above 1 cm.

Fig. 1.—Coronal section of right frontal lobe, with cavum septi pellucidi on the left, showing cavities in the subependymal glia with separation of the ependyma.

Microscopic Examination.—Sections of the brain were stained with hematoxylin and eosin and van Gieson, and with iron hematoxylin for myelin sheaths and neuroglial fibers.

Sections were examined from representative areas of the cortex and basal ganglia, from serial coronal blocks of the hypothalamus, and from sagittal and horizontal blocks of the brain stem.

Brain stem and hypothalamus: Roughly corresponding to the abnormal area observed macroscopically were numerous blood vessels lying in a glial matrix (Figs. 2, 3, and 4). This glia, although often fibrillary about the vessels and beneath the ependyma, was in places highly cellular with large prominent nuclei, so that in section it was not unlike astrocytoma. Small areas containing numerous compound granular corpuscles and spaces between the glial cells, suggesting recent softening and edema, were found at the junction of aqueduct and third ventricle, and in the medulla. One small recent hemorrhage was present in the area of softening near the aqueduct (Figs. 5 and 6).

The extent of this abnormal tissue was determined as accurately as possible by numerous sections, and was as follows: The area most affected was at the junction of aqueduct and third ventricle; it did not, however, extend rostrally as far as the dorsomedian nucleus, but the substance of the posterior hypothalamic nuclei contained vascularized tissue, although the number of vessels here was not great, and both large and small nerve cells seemed normal. The mammillary bodies were entirely free of the tissue, which was limited on its inferior and lateral margins by the mammillothalamic tracts. No other nucleus of the hypothalamus showed any significant changes, although all were examined.

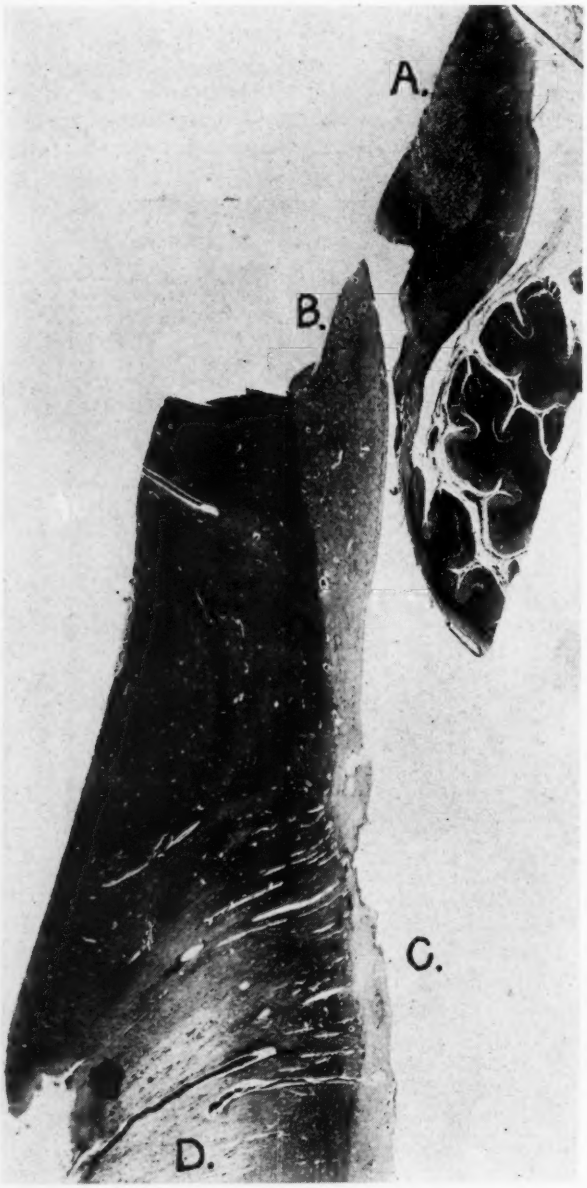

Fig. 2.—Sagittal section of lower part of the aqueduct and part of fourth ventricle. *A*, Vascularized and gliotic area beneath the quadrigeminal plate. *B*, The same anterior to the aqueduct and extending down under the floor of the ventricle *C*. *D*, The same in the pons (ironhematoxylin).

In the region of the aqueduct, especially at its rostral part, the vascularization and gliosis was most intense (Fig. 3), but the neighboring myelinated tracts and groups of nerve cells, some contained within the gliotic area, seemed healthy. On the right side, where the lesion was most extensive, numerous compound granular corpuscles were present indicating recent softening (Fig. 6). The vascularized area surrounded the aqueduct, extending caudally under the ependyma of the fourth ventricle on the right side and toward its right lateral recess.

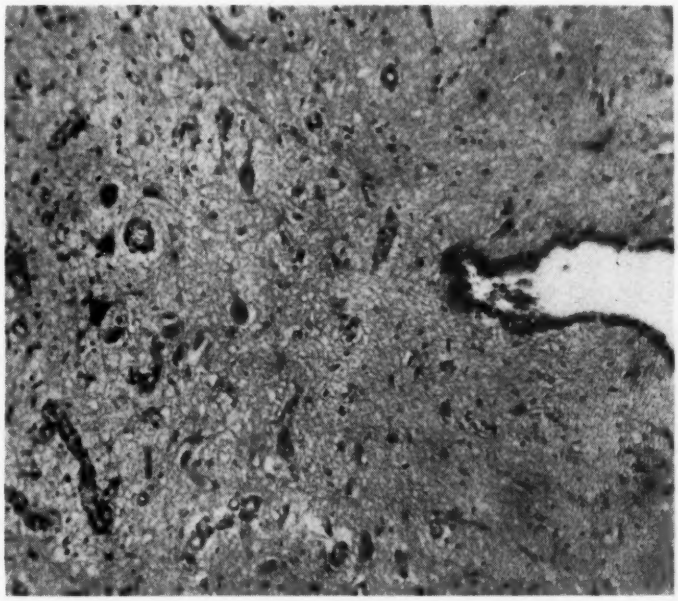

Fig. 3.—Gliotic area with new vessels near upper part of aqueduct (hematoxylin and eosin. ×100).

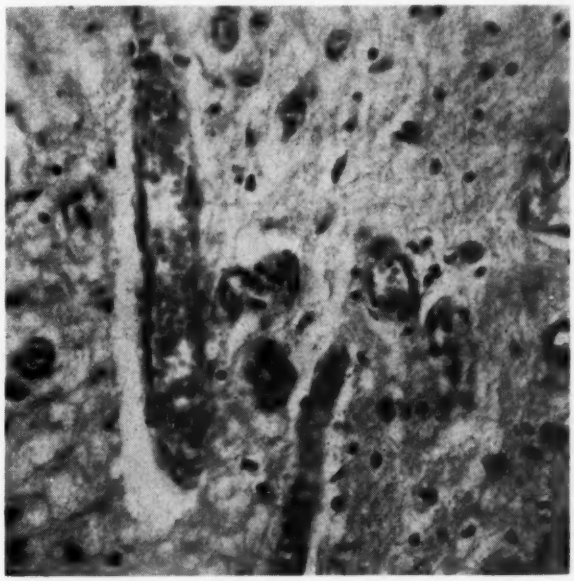

Fig. 4.—The same section as that shown in Fig. 3 revealing new vessels (×240).

The left side of the floor was little involved. Below the level of the pons a large area of gliosis and vascularization was present toward the midline just posterior to the inferior olives and involving both sides. A similar area, but containing many compound granular corpuscles, and therefore probably indicating recent softening, lay inferior to it in the medulla on the right side; it was probably continuous with the higher area. At this level the ependyma at the inferior angle of the ventricle was granular and gliotic over a small area not reaching the midline, just posterior and lateral to the hypoglossal nucleus.

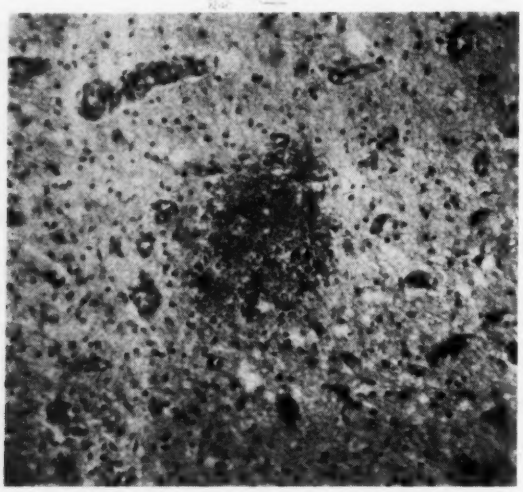

Fig. 5.—Hemorrhage in a small area of softening in the gliotic area near the upper part of the aqueduct (hematoxylin and eosin, ×90).

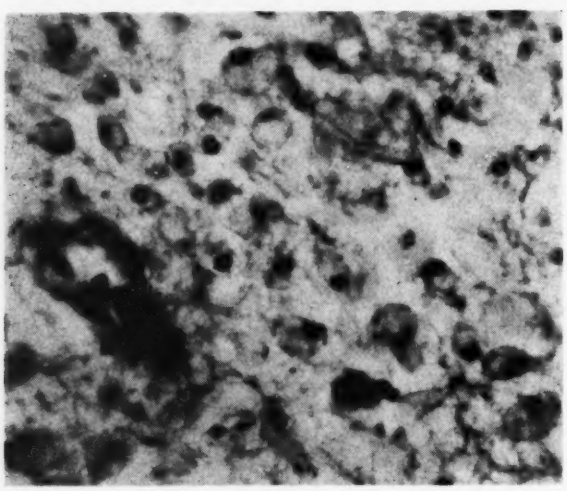

Fig. 6.—The same area of softening as that shown in Fig. 5, with compound granular corpuscles (hematoxylin and eosin, ×375).

The cerebral cortex appeared normal. There was, however, an increase in vessels and in neuroglial nuclei in the white matter of the hemispheres and in the basal ganglia. The abnormal area in the left external capsule proved to be a well-defined area of gliosis and vascularization with strands of connective tissue (Fig. 7). There was marked gliosis about the vessels and an increase in oligodendroglia in the neighboring brain. No compound granular corpuscles were observed.

The cerebellum seemed normal and contained no new vessels or areas of gliosis.

The ependyma of the cerebral ventricles showed occasional small microscopic granules of gliosis. Almost everywhere the subependymal glia was unduly thick. The cavities noted macroscopically were entirely within the glia, rough-walled, and did not contain blood pigment. The walls of the cavum septi pellucidi and Vergae were composed of similar tissue (Fig. 8). The subependymal glia often seemed unduly vascular, and in places the glial nuclei were so

Fig. 7.—Margin of gliotic area in left external capsule (hematoxylin and eosin, ×100).

Fig. 8.—Section near junction of septum pellucidum and ventricular wall; septum pellucidum to the left with ragged wall of cavum (hematoxylin and eosin, ×40).

large and numerous that the area could readily have been mistaken for glioma. Numerous nests of ependymal cells lay within the glial zone and extended for some distance along the inferior margin of the rostrum of the corpus callosum.

The subarachnoid space contained no abnormal cells, although the arachnoid was slightly thickened, possibly from hemorrhage at the time of injury.

DISCUSSION

Pathologic Features.—An opportunity to examine a brain many months after a severe head injury is not often given. In this case interest is added in that the injury was a closed one and there was no evidence at autopsy that the surface of the brain had suffered significant injury.

The following abnormal features seem worthy of record:

(1) The diffuse nature of the gliosis and vascularization, and in particular its severity at the junction of aqueduct and brain stem and about the aqueduct, and its extension under the floor of the fourth ventricle

(2) The marked participation of newly formed vessels in the reparative process along with gliosis

(3) The changes in the subependymal glial zone

(4) The presence of a few small areas containing compound granular corpuscles, suggesting recent softening

(5) The presence of a well-formed combined cavity derived from a combination of cavum septi pellucidi, and cavum Vergae

(1) The nature of the changes in the brain resulting from head injury are well known, and in particular those found in patients who died soon after trauma. Injuries of cortex and of the meninges need not here concern us, as they were not visible in this patient. There was, however, unquestionable evidence of widespread deep changes markedly involving brain stem, posterior hypothalamus, and the region adjacent to the left insula, although elsewhere present to a minor degree.

Their histologic structure was one of new vessel formation (Figs. 2 and 3) and gliosis. Such a reaction of the brain to injury is well known, new vessels being derived from those normally present, and glial cells with fibrils from the multiplication with fibril formation of classical neuroglia. Changes in vessels and neuroglia can be observed as early as the third and fourth days after injury.[3, 4] The reaction of all types of neuroglia to trauma has been well studied by Rand and Courville[4b] in early fatal cases, and the important effects of hemorrhage in producing tissue reaction have been emphasized.

However, in the late example here described there is no conclusive evidence of past hemorrhage, although it probably occurred. Gliosis and vascularization being widespread and sometimes well defined, it concerns us to know what lesions with this distribution and definition might legitimately be considered as their precursor.

Multiple hemorrhages, often of small size, have been commonly described in injury of the brain in distribution closely resembling that of the lesions under consideration, their first description in the brain and brain stem being usually attributed to Richard Bright in 1831.[5] Later they were reported in human beings and in traumatized animals by Duret in 1878,[6] and since by many others. Their common sites are the white matter of the cerebral hemispheres, brain stem, and upper cervical cord, although found in many other

positions. The gray matter of the cortex is rarely affected. It is to be observed that the zones of gliosis and vascularization in this case have appeared in those sites commonly affected by petechial hemorrhages, as for instance the region about the aqueduct, and the floor of the fourth ventricle, and the deeper part of the pons. They do not, however, always appear here experimentally, for Denny-Brown and Russell,[7] in extensive experiments on cats, found them most commonly in the gray matter and under the pia of the upper segments of the spinal cord.

The origin of these hemorrhages has been attributed to the force of the injury transmitted directly to the vessels, with fracture of their walls; to injury of the walls from the driving in of cerebrospinal fluid along the perivascular spaces; or to the concussive effect of injury on the vessel, with subsequent vascular dilatation, stasis of circulation, thrombosis, and anoxemia, followed by destruction of the wall. However, cases have been reported in which fresh petechiae have been observed days or weeks after injury, some of which certainly have resulted from fat embolism, but the origin of others has been uncertain and the theory of capillary stasis has again been invoked for their explanation. One small hemorrhage of recent origin in an area of softening was noted in this case (Fig. 5).

Furthermore, injury to the deeper parts of the brain, brain stem, and cord may occur without such overt evidence of injury as hemorrhage. This is clear from those cases of death attributed to concussion, where there was no visible appearance of local injury, although this must have occurred. It is also shown by areas of softening deep in the brain substance of certain acute fatal cases, and may be assumed from the widespread gliosis observed in other patients who died some time after injury. This occurrence of softening without hemorrhage is clearly seen in the patient (Case 1) reported by Winkelman and Eckel,[3] who died four days after injury. The cerebellum contained many areas of softening, with early vascularization, the microphotograph somewhat resembling those of the case here reported.

From such injuries, as well as from overt bleeding, gliosis may result, changes in the neuroglia often being visible within a few days. That generalized edema is uncommon, but local edema about areas of traumatized tissue common, has been emphasized by Greenfield[8]; in his opinion it may cause considerable demyelination with atrophy of tissue. Areas of gliosis observed later after injury, particularly if associated with paling of the white matter as shown by myelin sheath staining, may represent areas previously affected by such edema.

Applying these observations to this case it is likely that the well-defined gliotic areas in the brain stem have resulted from small hemorrhages. Although such hemorrhages may later produce cysts, large or small, with gliotic margins,[4b] this does not seem necessarily to follow. Many hemorrhages follow tissue planes, such as are present along vessels or between tracts of ensheathed nerve fibers; these would result in elongated areas of gliosis and vascularization without cyst formation, such as can be seen in the brain stem of this case, particularly in the subependymal zone.

The more diffuse gliosis and vascularization found in the white matter of the cerebral hemispheres could have resulted from somewhat widespread and less well-defined zones of degeneration and edema such as we have discussed. Nowhere was there clear evidence of old hemorrhage as shown by blood pigment accumulations, although occasionally microscopic cysts were observed in gliotic areas.

(2) The marked vascularization of the areas is notable. Prominent vascular endothelium has been observed even in cases of rapidly fatal head injury and evidence of new vessel formation has been observed within a few days of injury. Vascularization is a common feature in the repair of many brain lesions and may be a conspicuous feature in the reaction to many of the more malignant gliomas.

Where cortex and meninges have been injured, a highly vascularized scar can result, the composition of which has been studied by Penfield,[9] and Foerster and Penfield.[10] They have emphasized the role of this scar in the production of posttraumatic epilepsy, and that by its contraction, tension occurs between meninges and brain. The brain is thereby anchored at its area of attachment to the meninges and not permitted normal freedom of movement with respiration or with the filling of the cerebral vessels when the intracranial venous pressure is increased physiologically.

But not all patients with post-traumatic epilepsy show adhesions between cortex and meninges; at times the scar is within the brain without outward attachment yet clearly forms an epileptogenic focus in which glial and fibrous tissues are bound to new vessels. Exactly how such an area incites the epileptic attack is not clear, but it is plainly liable to changes in volume subsequent to passive engorgement for the new blood spaces usually form no muscular coat and are of the nature of capillaries. Furthermore, it is perhaps liable to certain degenerative changes; in two zones, at least, in this case there was evidence of recent lesions of a degenerative type, as shown by many compound granular corpuscles among the new vessels in the peri-aqueductal area of gliosis and in the medulla. The former lesion may have been of significance in the initiation of recent symptoms.

(3) Changes in the subependymal glia were well marked. The frequency of such changes, as well as those in the ependyma itself, can be seen in the large series of cases of fatal head injury studied by Rand and Courville.[4a] They observed edema of the subependymal zone, sometimes with extravasation of blood and vacuolization of the ependymal cells as a common feature, shortly after injury.

It is well known that small petechial hemorrhages of traumatic origin may be situated beneath the ependyma of the ventricles, and that at times the ventricular wall may be ruptured. This might well occur in that type of injury illustrated in the experiments of Denny-Brown and Russell[7] and called by them "acceleration concussion." Here the suddenness of the movement subsequent to the impact of the blood could result in a dragging away of the layer lining the ventricles, from the zone to which it is attached. It may have occurred

thus in this case; in many areas the ependyma was separated from the neighboring brain tissue or contained cavities which occasionally communicated with the ventricles. Cavitation was particularly marked about the anterior horns (Fig. 1), an area which Greenfield[8] found susceptible to injury when the posterior part of the head receives the blow. Furthermore, in this case there were areas in which small nodes of glial cells were present, occasionally the nuclei being of large size, the histologic appearance over a small area suggesting activity and resembling a moderately cellular glioma.

(4) The presence in this case of small areas containing compound granular corpuscles and one small hemorrhage has already been noted and commented upon. It is likely that such phenomena so long after injury have resulted from changes, such as thrombosis, in newly formed vessels.

It is important to emphasize that the late effects of cerebral trauma are by no means confined to the mechanical effects of scar contraction or to adhesions restricting normal movements, but that late softening and vascular changes may occur.

(5) The large central cavity, a combination of cavum septi pellucidi and cavum Vergae, could not be claimed reasonably as the result of injury. It is not proposed, therefore, to discuss the origin and significance of this condition here; it has been well described by Dandy,[11] who reported two similar cysts upon which he had operated subsequent to signs of organic brain disease, their sites being correctly localized by ventriculography. He, too, has fully traversed their historical discovery and anatomic features.

It is unlikely in this case that the abnormal cavity in any way contributed to the later clinical features, for at the time of autopsy it freely communicated with the ventricles. Earlier it may not have done so. Its presence indicates an abnormality of development, possibly affecting, too, the subependymal glia in general. It is, indeed, conceivable that the subependymal glia of this patient may have been unusually well developed at birth, for everywhere it was thick and contained more ependymal rests than are commonly seen. The septum pellucidum itself represents in part the subependymal glia zone, although containing some true nervous elements. Furthermore, there were occasional small subependymal granules which were not clearly attributable to the trauma and may have been congenital. The subependymal glia of this patient, indeed, may have been more susceptible to injury than is usual.

CLINICAL DISCUSSION

The chief clinical interest in the patient lies in the late occurrence of somnolence, with obesity, and excessive appetite and thirst, and in an unusual epilepsy conforming in part to the periventricular or diencephalic type. All of these late phenomena can be related to the lesions of hypothalamus, periaqueductal region, and brain stem with reasonable certainty.

The day has passed when proof need be shown that lesions of the hypothalamus can produce such symptoms as undue somnolence, abnormal thirst and appetite, and obesity; this has been amply shown by observations on such conditions as neoplasms of the brain base. Experiments on animals have also

confirmed the association and have shown that injuries in particular situations may produce one or another of the abnormalities of the "hypothalamic syndrome." However, one need not attempt to overload the individual nuclei of the hypothalamus with such restricted functions as the control of sleep, the vasomotor system, or body temperature, even if there is much evidence to show that certain nuclei may modify individual functions, mainly through their connections with hypophysis, for the supraoptic nucleus by its control of the antidiuretic hormone is related to water metabolism, its injury resulting in diabetes insipidus, and the ventromedial nucleus may control the diabetogenic hormone with the production of diabetes mellitus at its injury. Such a clear allocation of function has not been shown for the posterior nuclei, even though it may be generally stated that the posterior hypothalamic area plays some role in the coordination of some sympathetic reflexes, and the anterior a smaller part in coordination of parasympathetic reflexes.[12a]

The physiologic functions which were altered in this patient were those of sleep, appetite, thirst, and control of heart and respiration. Furthermore, the gain of weight suggested a metabolic change.

Excessive sleep has been frequently produced by lesions of the hypothalamus, through disease, or by animal experimentation. In animals, Beattie, Brow, and Long[13] found its production outstanding in those which survived section of the tracts between the anterior edge of the superior colliculus and the mammillary bodies. These results have been confirmed by experiments with the stereotaxic instrument in which it has been possible to destroy the gray matter surrounding the opening of the aqueduct into the third ventricle, with only a small amount of surrounding tissue.[12a] Ingram, Barris, and Ranson[14] found somnolence to be best produced by lesions between the mammillary bodies and the third nerve nucleus. As in both groups of experiments there need be no injury of the long motor or sensory tracts, they agree that destruction of the efferent hypothalamic connections is the essential feature.

How these lesions cause somnolence is as yet unproved. To some the sleep impulse is an active inborn process analogous to an instinct; it is something so vital to the organism and appearing in so many species, that it seems a mechanism must exist for its promotion. I have allied myself to this view[15] and am of the opinion that it may yet be shown that pathologic somnolence of the type being considered results from the physiologic release of an inborn mechanism, which thereby overacts. Others regard it likely that a mechanism for attention exists and that the lesions of the hypothalamus or thalamus prevent it exerting its control, body and brain thereby falling into a resting state through lack of rousing.

Whatever the correct explanation, it is certain that other changes in consciousness, such as catatonia, and trancelike states similar to, but not identical with, sleep may be encountered with pathologic lesions in this neighborhood. I have reviewed the subject with illustrative cases,[15] and more recently it has been reviewed by Alpers.[16a] That the loss of consciousness in concussion may be derived from a disturbance in such a mechanism is no new view; the idea

that concussion is produced through lesions of the brain stem is many years old, and recently the resemblance of the loss of consciousness to sleep has engaged the attention of Eden and Turner[17] and Jefferson.[18]

Furthermore, sleep is intimately bound up with physiologic effects of the vegetative type; it is not surprising, therefore, to find disorders of these functions with lesions of the hypothalamus which produce hypersomnia.

Obesity is also a common association of hypothalamic lesions, although as yet there is no certainty as to its mode of origin. It may occur with lesions of the hypophysis as well as of the hypothalamus. Excessive appetite and thirst are often its associates, and all, although not of necessity, are associated with hypersomnia. Large lesions of the tuberal region seem most likely to produce obesity experimentally.

It is of interest to note that in this case with all these symptoms present, the mammillary bodies were not involved, and of the hypothalamic nuclei, only the posterior group was involved, although both large and small nerve cells seemed normal histologically. However, as these cells lie in the course of the periventricular system of fibers and seem to be an important source of its origin, it is likely that involvement of the tract, rather than any particular group of nerve cells, as in animal experiments, is the essential lesion in hypersomnia. Indeed, it can be seen in this case that the zone of vascularization and gliosis in hypothalamus, mesencephalon, and pons closely followed the course of these fibers. The periventricular system is considered an efferent pathway from the hypothalamus carrying impulses from diverse hypothalamic nuclei to the brain stem and perhaps to the spinal cord.[12b, 16b] As the fibers are related to nuclei of the motor cranial nerves and of the reticular substance, it is likely that they are concerned with visceromotor functions. Therefore, it is not difficult to imagine that such functions could be disturbed with lesions of the hypothalamus and brain stem of the type discovered in this patient.

The reason for the late incidence of the hypothalamic symptoms is by no means clear. Why should such phenomena occur late, rather than early, when the lesions were presumably at their maximum? Is there evidence for progression in traumatic lesions of the cerebrum?

There is no easy explanation for these late phenomena. Should they require a paralytic lesion for their production, such was certainly present in the early stage of injury. It has already been noted that there was evidence in this patient of a recent lesion at the junction of aqueduct and third ventricle, as well as in the medulla; in the former lesion numerous compound granular corpuscles, one small recent hemorrhage, and some separation of glia cells suggesting edema were present on the right side.

If, however, in analogy with ordinary post-traumatic epilepsy, a focus of irritation is thought to be the initiator of the epilepsy, we can regard this same area of softening as an adequate cause or fall back on that explanation usually deemed sufficient for this condition, irritation from the contraction of connective tissue and glia in the process of repair, with alterations in volume of the new vascular bed. Either view might imply that the hypothalamic syndrome of

obesity, excessive appetite, and thirst with hypersomnia also arose from a constant or frequent irritative process, and the later epileptic condition with the respiratory and cardiovascular disturbance, raised temperature, disturbance of consciousness with excessive hunger was but the result of a greater and more widely spreading discharge.

Attacks of this type were described by Gowers[19] as vasovagal attacks, and by Wilson[1] as periventricular epilepsy. There was little satisfactory pathologic confirmation for these early cases, but Penfield's patient[2] who suffered attacks of restlessness, with thirst, a sensation of heat, vasomotor changes, sweating, lacrimation and salivation, pupillary changes, and sometimes exophthalmos, was found to have a tumor involving both foramina of Monro. McLean's case[20] was also of this type. With the attacks were emotional changes, dyspnea, oppression of the chest, tachycardia, hunger, and impulsive weeping; a large basal tumor was present. One of my patients (Case 6[15]) suffered from turns of mental distress with respiratory and vasomotor changes. He was found to have a pituitary tumor and was relieved by operation.

Nevertheless, small circumscribed lesions have not usually been found at autopsy. Even in the patient here described other lesions than those in the hypothalamus and brain stem were present; thus, besides widespread changes in the white matter and the basal ganglia, a large lesion was found in the left claustrum. That a hypothalamic lesion was present was thought possible, clinically, from earlier experience with basal tumors, and it seems highly probable that the lesion in brain stem and hypothalamus was the exciting cause of the late symptoms.

The hemiplegia early in the illness is presumed to have resulted from the large lesion in the left insular area.

SUMMARY

1. A boy, aged 8 years, although apparently recovering well from a severe head injury, developed, after thirteen months, voracious appetite, thirst, rapid gain of weight, and undue sleepiness.

2. One month later he developed attacks, with vasomotor and respiratory changes, and hunger, in which he usually became sleepy or semiconscious. Sometimes clonic contractions of the limbs were present. During their course he died.

3. At autopsy a prominent area of gliosis and new vessel formation surrounded the aqueduct, extending well down into the brain stem. Lesser changes were present elsewhere, although a gliotic area was present in the left internal capsule. There were a few areas of recent softening.

4. The hypothalamic nuclei were not affected, the lesion appearing chiefly to affect the zone of the periventricular fibers.

5. There was considerable cavity formation in the subependymal glia of the ventricles, and a large combined cavum septi pellucidi and cavum Vergae was present.

6. The relation of the pathologic condition to the injury and its anatomic and clinical implications are briefly discussed.

I am indebted to Dr. H. C. Trumble, with whom I saw this patient, for permission to study and describe the brain; and to Professor R. Willis and to Dr. H. Sissons for the photographs.

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CONTRALATERAL, IPSILATERAL, AND BILATERAL REPRESENTATION OF CUTANEOUS RECEPTORS IN SOMATIC AREAS I AND II OF THE CEREBRAL CORTEX OF PIG, SHEEP, AND OTHER MAMMALS

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INTRODUCTION

STUDY of central afferent systems by recording electrical potential changes evoked on physiologic stimulation of peripheral receptors or by electrical excitation of peripheral nerve fibers, as distinct from observation of spontaneous electrical rhythms or of potential changes induced by local applications of strychnine,[23] is now a well-established anatomicophysiologic procedure. It has been successfully employed on the cerebral cortex for investigating localization in the somatic,[1, 2, 4, 10, 11, 28-30, 37, 38, 54, 55, 57, 58, 60] the visual,[29, 35, 36, 39, 45-48] the olfactory,[3, 8, 26, 43] and the auditory systems.[6, 7, 14, 15, 34, 49, 52, 59] In the cerebellum it has revealed that not only vestibular[21] and proprioceptive impulses[22] but also visual,[44] auditory,[44] and tactile[5, 22, 44] messages are received by this organ in well-organized systematic fashion.

With animals under satisfactory barbiturate anesthesia, careful research reveals in great detail the manner in which the various receptor surfaces are represented in the central structures and makes possible comparative functional and morphologic investigations unattainable with any other method. It is becoming evident that the evoked potential technique permits identification of certain homologous cortical areas more definitely than does the cytoarchitectonic method and in addition it makes possible detailed comparison of relationships within homologous areas. Clearly, combination of electrical technique with classical anatomic and physiologic procedures will result in great refinement of our knowledge of functional organization within the nervous system.

The time is now at hand for the study of cortical potentials evoked in man at neurosurgical operations. It is particularly important, therefore, in order that significant answers be secured that general principles of cortical organization be defined clearly by comparative studies on lower forms.

Until now it has been customary to think of each of the main afferent systems as having a single primary pathway into the cerebral cortex. The present study and earlier observations on cat,[1, 54] dog,[54] monkey,[54, 55] and rabbit[60] show that the somatic system has two distinct afferent areas with separate pathways.[60] The same has been found also for the visual[45, 48] and the auditory[49, 59] systems. Duality, therefore, appears to be a general principle of cortical organization.*

*In discussing these dual systems we shall refer to them as somatic areas I and II, visual areas I and II, and auditory areas I and II. This terminology was chosen because it carries no anatomic or functional implications and because it can be applied to each of the three systems. Moreover, area I corresponds to what has been considered until now the primary receptive area of each system. Area II in each case was "second" with respect to area I in time of discovery.

In the present study we have undertaken to answer certain questions raised by the recent observations of Adrian[4] on the ungulate brain. Previous studies on monkey,[55, 57] cat,[30, 54] dog,[54] and rabbit[60] had led us to the generalization that in each of these species the somatic receiving areas are fundamentally similar. Homologous subdivisions of the "postcentral" area (somatic area I) are identifiable in each animal. The area differs from species to species, not basically but, in the relative development of its various subdivisions, apparently in accord with the specialization of the corresponding peripheral receptor surfaces in individual species. In somatic area I all responses had been found to be strictly contralateral, with the exception of a part of the face area of the monkey.[57] Besides the "postcentral" area we have found in each species an area (somatic area II) homologous with Adrian's[1, 2] "second" somatic receiving area of the cat. In rabbit, cat, and dog this area is situated lateral to somatic area I and anterior to the auditory areas (Fig. 6, A and B); in monkey it lies chiefly on the dorsal wall of the sylvian fissure in similar relationship to somatic area I of the postcentral gyrus and to the auditory areas (Fig. 6, C). Our studies have shown that in somatic area II both halves of the cutaneous surface are represented in each cerebral cortex. Contralateral responses are approximately twice as large as corresponding ipsilateral ones. The apical portions of the body (digits, snout, and tail) give largest responses on stimulation, and under deep anesthesia the apices of the contralateral half of the body alone may activate the cortex. There is spatial differentiation within somatic area II and this increases from rabbit to monkey but it is much less than the increasing differentiation within somatic area I. It does not reflect the specialization of receptor surfaces as does somatic I.

Adrian's[4] study on ungulates indicated that from the face only receptors in lips and snout sent impulses to the cortex. In sheep and goat these impulses were ipsilateral while in pig and horse they were contralateral. In addition, responses to stimulation of the contralateral forelimb and sometimes of the contralateral hindlimb were observed in the anterior ectosylvian gyrus of goat, sheep, and horse. No responses from the limbs were evoked in pig and in none of the species was any response to limb stimulation detected in areas possibly homologous with postcentral gyrus.

To relate these findings on ungulates to results on other species, observations were made on one sheep and seven pigs (the numbers being dictated by supply). We soon found in sheep and in pig both the contralateral face area of pig and horse and the ipsilateral face area of sheep and goat. Most of the previously defined characteristics of somatic area II were established also for sheep and pig, and in addition the arm subdivision of somatic area I was located in pig. (It was not looked for in sheep.) A hindlimb subdivision of somatic I was not found but its probable locus was suggested.

With discovery of the ipsilateral face area in pig and with the existing evidence for a bilateral face area in monkey, search was made for a homologous area in cat and dog. This was found in both species, thereby establishing for all animals studied a basic plan of cortical organization. Data from the literature will be cited with a view to relating some of the findings to man.

MATERIALS AND METHODS

The animals used in this study consisted of three Yorkshire and four Hampshire pigs, aged 5 to 8 months, one sheep, aged 4 months, two cats and one dog.

All animals were anesthetized with pentobarbital sodium, administered intraperitoneally, except for four of the pigs, which were given a combination of pentobarbital and chloralose. Initial dosage of pentobarbital was 42 mg. per kilogram. Anesthesia was maintained as evenly as possible by periodic injections of additional amounts (every twenty minutes in case of pig). In the pigs the maintenance dose of pentobarbital was found to increase with weight from 5.5 mg. per kilogram per hour for a pig weighing 9.5 kilograms to 15.0 mg. per kilogram per hour for one weighing 17.4 kilograms. Chloralose in the amounts used (11 to 33 mg. per kilogram per hour, approximately) had little if any anesthetic effect; the maintenance dose of pentobarbital was the same for animals of equal size whether or not chloralose was given.

Chloralose was employed to facilitate transmission of afferent impulses to the cortex. This seemed clearly to be its effect because cortical responses were more easily evoked in the animals to which this drug was given and it was only in these animals that light tactile stimuli were adequate. Pigs under pentobarbital-chloralose anesthesia reacted more like cats under pentobarbital alone and did not show the convulsive features exhibited by cats under chloralose.

The pigs and the sheep salivated profusely. To avoid difficulty with respiration it was necessary to cannulate the trachea and to ligate the trachea above the cannula.

In exposing the brain the soft tissues were removed from the skull with a Davis-Bovie electrosurgical unit. The skull was then trephined and the bone removed with rongeurs over the entire dorsolateral surface of one hemisphere. In three of the pigs both hemispheres were exposed in the course of the experiment. In certain instances cortex on one wall of a fissure was examined after the cortex of the opposing wall was carefully removed by suction.

Before exposure of the brain the head was immobilized in a holding apparatus. Later a movable electrode carrier was attached to the headholder in such a way that the exploring electrode (made of steel tubing and a core of No. 50 cotton thread moistened with saline solution) could be moved over the cortex in millimeter steps along rectangular coordinates. The "indifferent" electrode was clipped to the scalp, usually in the frontal region. To prevent drying, the exposed cortex was covered with mineral oil.

The methods of stimulation and the apparatus for amplifying and recording the cortical electrical responses have been described elsewhere.[37, 38, 57, 59] The cutaneous stimulator consisted of a lever rigidly attached to the moving armature of an electromagnetic device whose coils were energized by a pulse 3 to 5 milliseconds in duration. A regular, quick, to-and-fro movement resulted, which produced at the end of the lever a small displacement lasting a few milliseconds. For tactile stimulation a camel's-hair brush was attached to the lever's end. For stronger stimulation the lever itself was used or a wire was mounted at right angles to the end of the bar. The action of the stimulator was synchronized with the sweep of the cathode ray tube, thus making it possible to visualize and to photograph the response produced by each stimulation.

RESULTS

Fig. 1 is a composite diagram illustrating the results obtained in the seven pigs. The brain is not oriented in the conventional manner, but is inverted, as are the other brain drawings in this paper. This arrangement was chosen in order to place the head uppermost in the figures and to put the feet down. We believe this aids in visualizing the plan of cortical organization, especially with respect to details within the face area (see Fig. 5), since the parts represented fall into position in the diagram in essentially the same relationships as exist in the actual face. The figure shows (1) the auditory area (*AUD.*), (2) somatic area II with its face (F_{II}), arm (A_{II}) and leg (L_{II}) subdivisions, (3) the face (F_I) subdivision of somatic area I (that is, the contralateral snout area of Adrian),[4] (4) the ipsilateral face area (*IPSI.*), which is homologous with Adrian's ipsilateral face area for sheep and goat, (5) the arm subdivision (A_I) of somatic area I and (6) the probable location of the leg subdivision (L_I) of somatic I on the mesial aspect of the hemisphere.

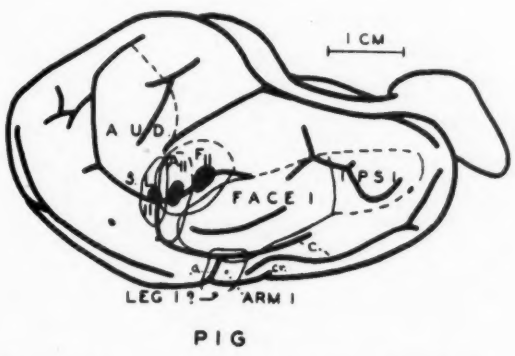

Fig. 1.—Composite diagram summarizing results obtained on seven pigs. The thin continuous lines mark boundaries determined by the experiments; the broken lines are provisional. The solid black areas represent foci of largest response to stimulation of face (F_{II}), arm (A_{II}) and leg (L_{II}) subdivisions of somatic II. For further description see text. Labeled fissures in Figs. 1 and 2 are: *a.*, ansate; *c.*, coronal; *cr.*, cruciate; *s.*, suprasylvian.

Data for Fig. 1 were obtained on examination of ten hemispheres in seven pigs. Not all the areas illustrated were examined in each hemisphere. Observations were made on somatic area II in nine, on contralateral face area of somatic I in nine, on ipsilateral face area in four, and on the arm area of somatic I in nine. A hindlimb subdivision of somatic I was searched for on the dorsal surface of the hemisphere in most of the animals but it was not found there. The supply of animals gave out before a satisfactory examination of the mesial surface could be made, but since arm area I extends to the midline, it is likely that leg area I is to be found on the mesial aspect of the hemisphere.

Fig. 2 illustrates our findings on the one sheep studied. In this experiment it was possible to locate (1) the auditory area (*AUD.*), (2) somatic area II with face (F_{II}), arm (A_{II}) and leg (L_{II}) subdivisions, (3) Adrian's ipsilateral face area (*IPSI.*) and (4) a contralateral face area (F_I) homologous with the contralateral face area of the pig. Arm and leg subdivisions of somatic I were not looked for.

Pigs and sheep proved much more difficult animals in which to study evoked potentials than have other animals with which we have had experience. The barbiturates, which have been the anesthetic of choice for evoked potential studies, were found to depress transmission in the afferent systems of these ungulates before they quieted the animals generally or suppressed the spontaneous brain wave activity. With prolongation of the anesthesia, however, transmission improved and reliable results were secured, but the difficulties necessarily limited the amount of information which could be obtained from each animal. In the later experiments pentobarbital-chloralose anesthesia was found to give a preparation more like cat under pentobarbital alone and from these animals more data were collected. In spite of these limitations the results obtained showed excellent agreement when data from all animals were assembled. We are confident, therefore, that the positive findings will stand. Our data are also in excellent agreement with the positive findings reported by Adrian.[4]

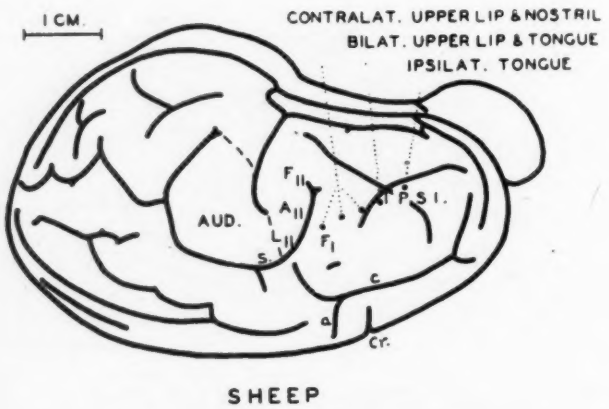

Fig. 2.—For description see text.

Fig. 3 shows the character of the electrical potential changes produced in the cortex of the pig by localized mechanical stimulation of cutaneous receptors. Responses *A* and *B* were recorded from a point in the arm subdivision of somatic area I (point *A* in Fig. 5, C). Response *A* was evoked by stimulating with a camel's-hair brush the skin over the anterior aspect of the contralateral forelimb just above the interdigital web. Response *B* resulted from stimulation of the same spot with a wire mounted on the lever arm of the stimulator. Both responses are initially surface positive but the time to maximum is shorter in *B*. Records *E*, *F*, and *G* were obtained in another animal from a point in the arm subdivision of somatic area II (point *E*, Fig. 4, B). *E* was produced by stimulating the skin above the interdigital web of the contralateral forelimb and a camel's-hair brush. *F* resulted from stimulation of the same cutaneous spot with the wire and *G* was evoked by wire stimulation of the corresponding point on the ipsilateral forelimb. It can be seen that the latencies, the times to maximum, and the durations of the surface positive waves of the somatic I responses are shorter than those of the somatic II responses. The differences in

form are due, in part at least, to differences in the time constants of the interstage couplings of the amplifier (see *D* and *I*). The longer times to maximum of responses *A* and *E* may be attributable to the less abrupt onset of stimulation by the brush as compared with the wire. Of the somatic II responses the ipsilateral has a longer latency than the contralateral. Usually the ipsilateral response was not more than one-half as large as the contralateral. In the records shown they are nearly equal in amplitude. Some observed values for the times to maximum were as follows: auditory responses, 22 milliseconds; contralateral face II, 22 to 32 milliseconds; contralateral arm II, 25 to 36 milliseconds; contralateral leg II, 45 to 48 milliseconds; contralateral and ipsilateral face I, 15 to 24 milliseconds; arm I, 20 to 22 milliseconds.

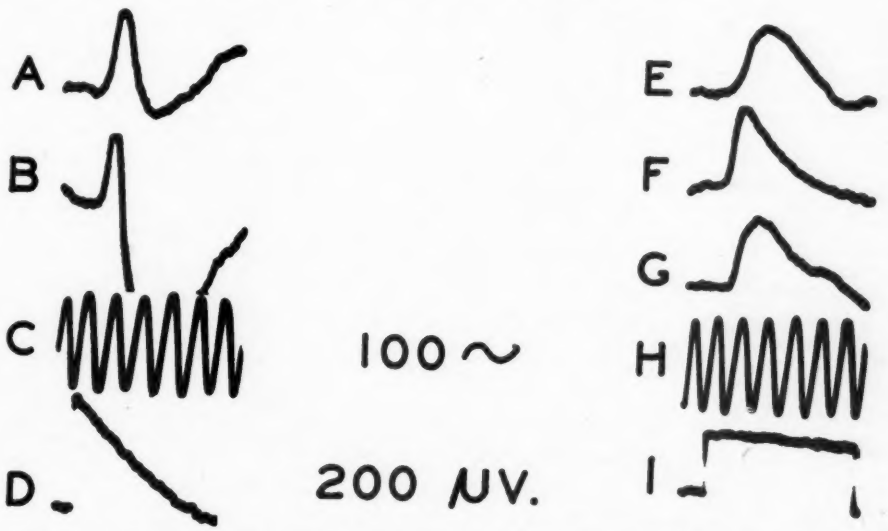

Fig. 3.—Surface positive cortical potentials evoked by stimulation of cutaneous receptors in the forefoot. See text. Records *C* and *H* are time signals and *D* and *I* are calibration potentials.

In working out relations of cortex to cutaneous surface, two types of experimental procedure may be employed. In one method a fixed point on the cutaneous surface is stimulated mechanically at approximately one-second intervals and the cortex is examined by moving the exploring electrode in 2 mm. steps along rectangular coordinates. In this way the cortical areas of response corresponding to particular cutaneous spots stimulated may be defined (Marshall, Woolsey, and Bard,[38] 1941). In the second method the cortex is examined point by point and for each cortical point studied the entire body surface is explored with the tactile stimulator to determine whether or not any peripheral area sends impulses to that cortical point. When a cutaneous area related to a cortical point is located, its borders are carefully delimited and the amplitudes of cortical potentials evoked by stimulation of various portions of the area are noted. This procedure defines the cutaneous areas related to corresponding cortical points and locates the peripheral locus giving largest responses at each cortical point (Woolsey, Marshall, and Bard,[57] 1942). The two methods reveal different aspects of cortical organization and with sufficient

observations the results of either procedure may be derived from data obtained with the other.

Fig. 4, A shows the responses evoked in somatic area I (Fig. 4, B) by light pressure stimulation of a fixed point on the dorsum of the contralateral forelimb. The cortical area defined is fairly extensive but it must also extend into the adjacent fissures. The largest responses of shortest latency, however, are fairly well localized. If one delimits the cortical response areas for a number of spatially separated cutaneous spots on the contralateral forelimb, the cortical areas are found to overlap one another greatly but the locus of the maximal

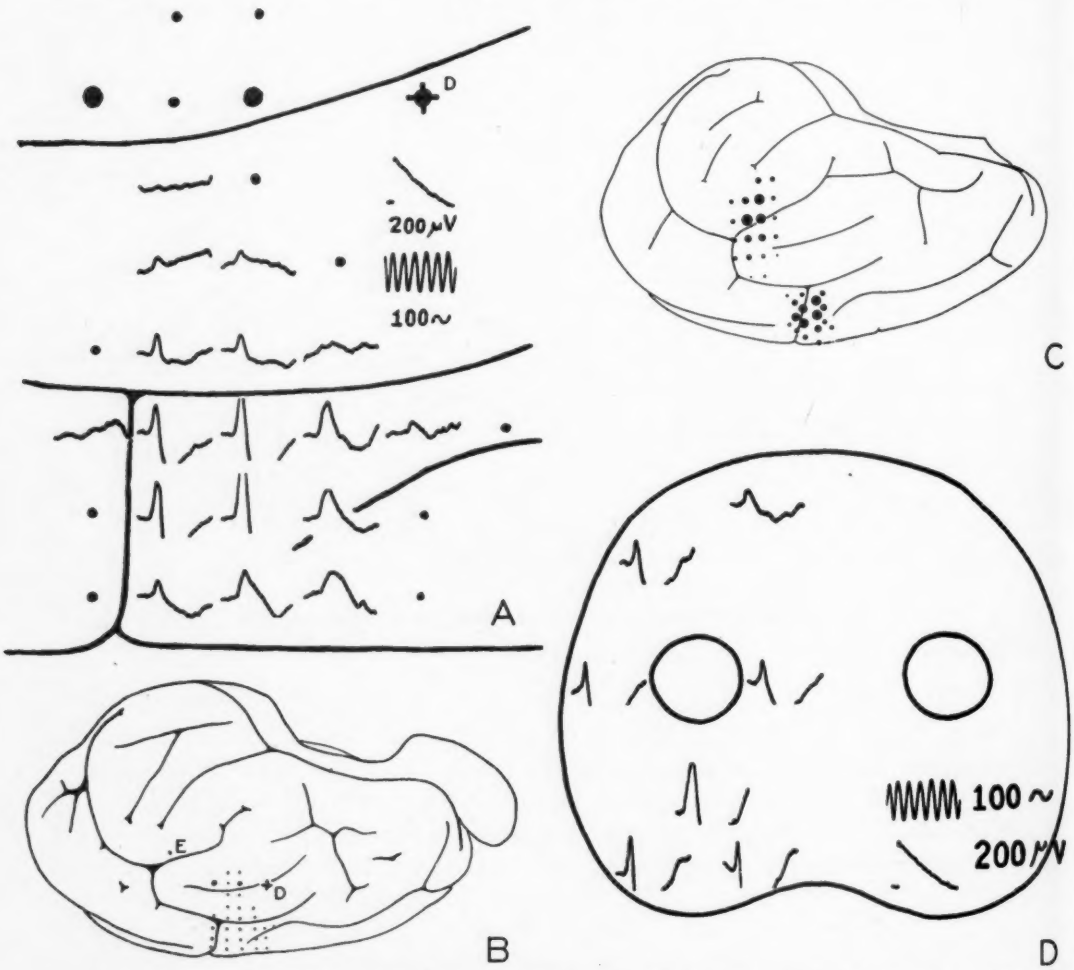

Fig. 4.—A, Cortical potentials evoked in arm subdivision of somatic area I by light tactile stimulation of the skin of the contralateral forefoot just above the interdigital web, volar aspect. Dots indicate points which yielded no response.

B, Drawing, after a photograph, of the brain from which potentials in A were recorded. The small dots represent cortical points examined and correspond to dots and records in A. The large dots are points in the face area for which skin areas activating the points were defined (see Fig. 5). From point *D* the responses shown in D were recorded. The dots in B are 2 mm. apart along rectangular coordinate.

C, Fields of response in somatic areas I and II to stimulation of contralateral forefoot. The diameter of each dot is proportional to the amplitude of the response at the point.

D, Responses evoked at point *D* in B on stimulation of various spots on the snout with a camel's-hair brush.

response is at a slightly different point on the cortex for each cutaneous spot. This arrangement no doubt provides not only a basis for local sign but also a mechanism for cortical integrations.

In Fig. 4, C the cortical areas of response to stimulation of a spot on the dorsum of the contralateral forelimb just above the interdigital web are indicated by dots whose diameters are proportional to the amplitudes of the responses observed at the corresponding points. There are two response areas. One is in somatic area I and the other is in somatic area II. In this experiment the response area in somatic I was found to extend into the ansate sulcus posterior to it and the response area in somatic II was followed to the bottom of the suprasylvian sulcus which cuts through the area. It should be noted that in addition to these two areas in the contralateral hemisphere, responses to the same stimulations must also have occurred in somatic area II of the ipsilateral hemisphere.

Data obtained with the second method are shown in Fig. 4, D. The exploring electrode was placed on a point in the contralateral face area (point *D* in Fig. 4, B). The cutaneous area activating the point was then found to be the contralateral half of the snout. Light tactile stimulation alone was adequate and with this no responses were evoked on ipsilateral stimulation. Largest responses with shortest latencies were obtained on stimulating the snout below the nostril. The records shown in Fig. 4, D represent the responses evoked at the cortical point when the part of the snout on which the record is mounted was stimulated. For systematic mapping of the cortex this second method produces more useful data in a given period of time than does the first method. Data of the type shown in Fig. 4, D may be transformed into small figures for assembling into charts by representing in black the cutaneous area producing largest responses and by lining the surrounding portions which yield smaller responses. In this way the figurines composing Fig. 5 were constructed. Each figurine was then mounted on a diagram of the brain in a position corresponding to the point on the cortex to which the cutaneous area illustrated sent impulses.

Fig. 5 is a composite chart made from data collected in four experiments. The figurines from each experiment were fitted together by superimposing drawings of the individual brains and matching the corresponding fissures as closely as possible. Fig. 5, C, which is also a composite drawing, shows the relations of the charted areas to the gross morphology of the brain. It must be emphasized that the results shown represent an incomplete map of the receiving areas of the pig. This is evident if one compares the data for arm subdivision of somatic area I, as shown in Fig. 4, A and in Fig. 5. But other portions of the map are also deficient, for example, the ventrolateral portion of somatic area II and parts of the contralateral and ipsilateral face subdivisions of somatic I. Large portions of the receiving areas must also exist in the various fissures. These have been explored only in part. It can be seen from Fig. 5 that the contralateral face area includes the medial bank of the coronal sulcus. It, therefore, must also occupy the lateral bank of the sulcus. The suprasylvian sulcus is nearly 1 cm. deep. Somatic area II descends to the bottom of this sulcus on its lateral bank, as we know from direct examination, but the results shown in Fig. 4, C indicate that it must cover the medial bank as well. In two of the

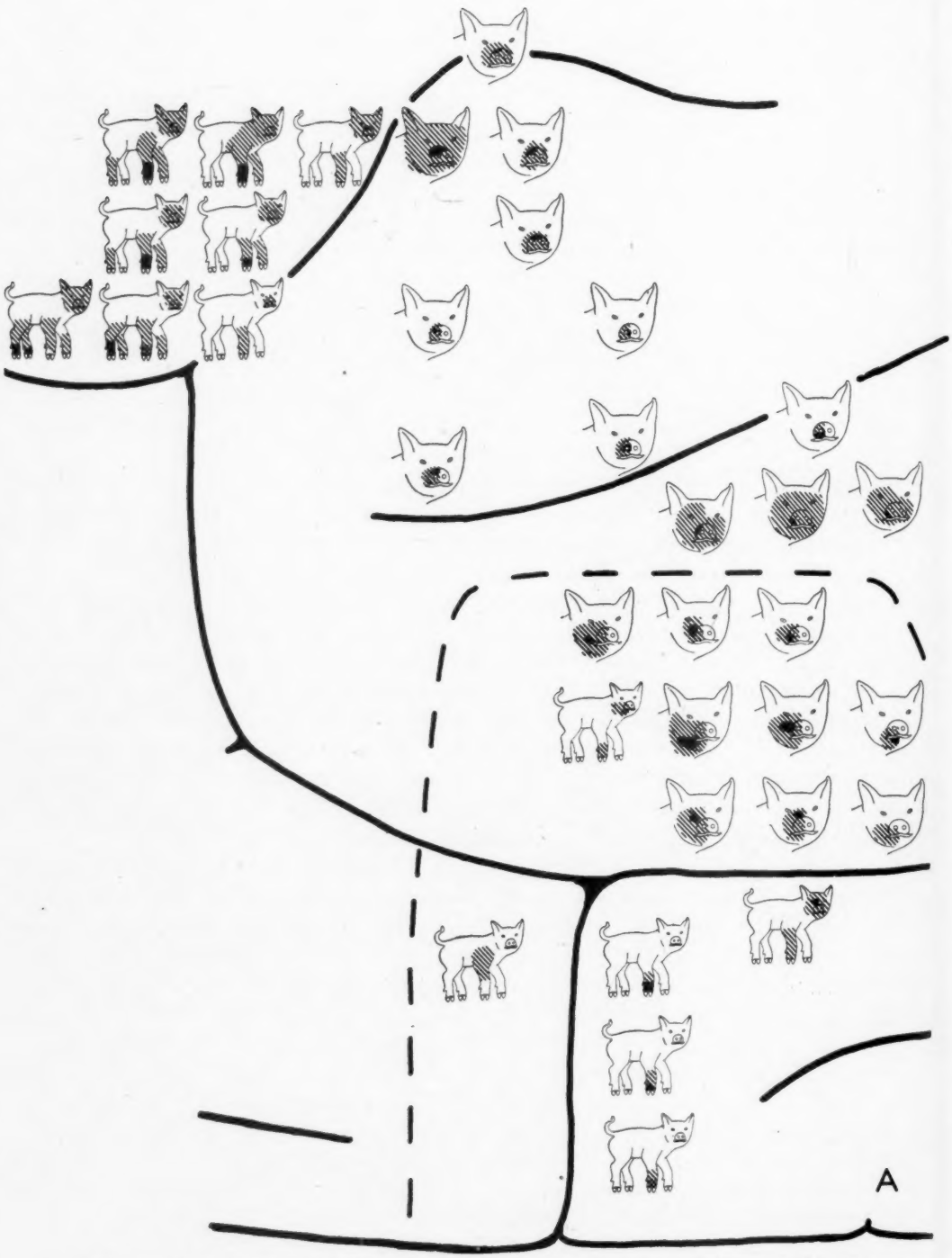

Fig. 5.—Each figurine in this chart shows the cutaneous area which sent impulses to the cortical point on which the figurine is centered. Largest responses were evoked by stimulating the area outlined in solid black. The two parts of the figure, A and B, correspond to the regions outlined by the dotted lines in C. The broken line in A delimits cortex on the medial bank of the coronal sulcus and the anterior bank of the ansate, exposed by removing opposing banks. Points on these banks are indicated by elongated dots.

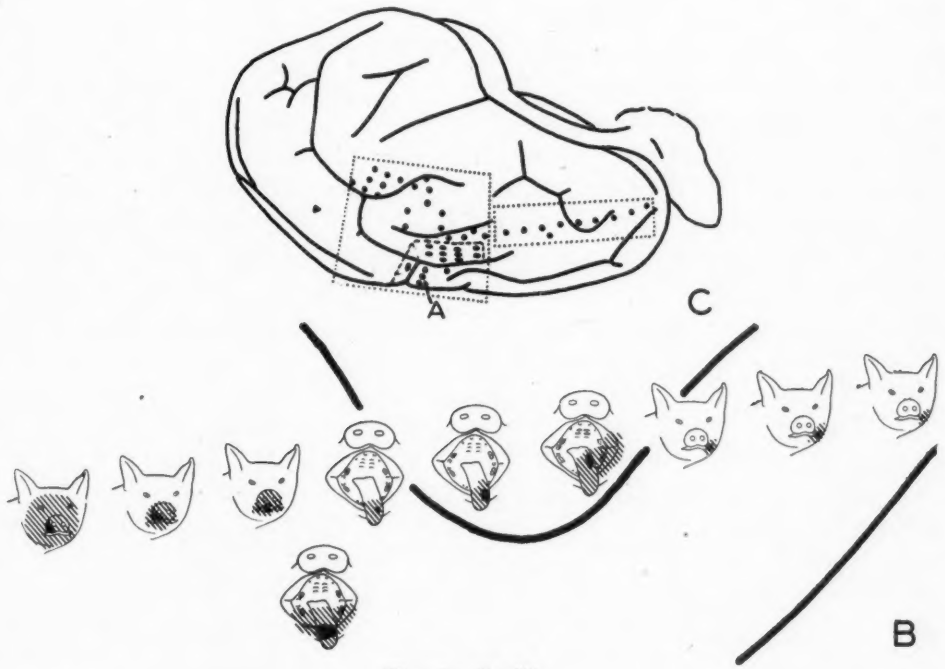

Fig. 5.—Cont'd.

pigs studied the hindlimb portion of somatic area II was entirely within the suprasylvian sulcus and in one it extended medial to the suprasylvian, posterior to the coronal (see Fig. 1).

Attention is called to several features of Fig. 5: (1) The relations of the cutaneous surface to the cortex are bilateral in somatic area II. This is quite clear with respect to the limbs where the possibility of mechanical transmission of the stimulus from one side to the other was eliminated. Controls to minimize this factor in the snout indicated that here too the bilateral relations were real. As the shading in the figures indicates, the contralateral responses characteristically were larger than the ipsilateral responses. (2) There is considerable overlapping of somatic II face, arm, and leg subdivisions, but the maximal response at each point is from either face, arm, or leg. (3) Somatic area II receives impulses principally from the apices of the limbs and from the upper part of the snout. Few responses were evoked by stimulation of the trunk, but in some pigs large potentials were observed on stimulation of the tail. (4) At several points within the contralateral face area the figurines show that the peripheral areas for these points extend to both sides of the body. Bilaterality here may be artefactual because of the strong stimuli employed. In any case, the responses were much larger contralaterally for all points in this portion of the face area. Moreover, no ipsilateral effects were ever produced in this area by brush stimulation alone. (5) Within the contralateral face area the pattern of representation on the free surface of the cortex is the same as that described by Adrian.[4] The lower part of the snout is represented rostrally in the area, the

upper part caudally. Maximal responses for points on the free surface of the cortex were always produced by stimulation of some portion of the snout. To obtain maximal responses from cutaneous areas not on the snout it was necessary to examine cortex buried within the coronal sulcus. On the medial bank of this sulcus maximal responses were produced by stimulation of the side of the face, the angle of the mouth, and the chin. (6) The ipsilateral face area receives impulses chiefly from the lower lip, the tongue, the floor of the mouth, the hard palate (not shown in figure), and the side of the face near the angle of the mouth. The transition from contralateral to ipsilateral face areas is marked by a shift of the peripheral area from central portions of the lips to the tongue. This was also found in the sheep (Fig. 2) and in other species studied. (7)

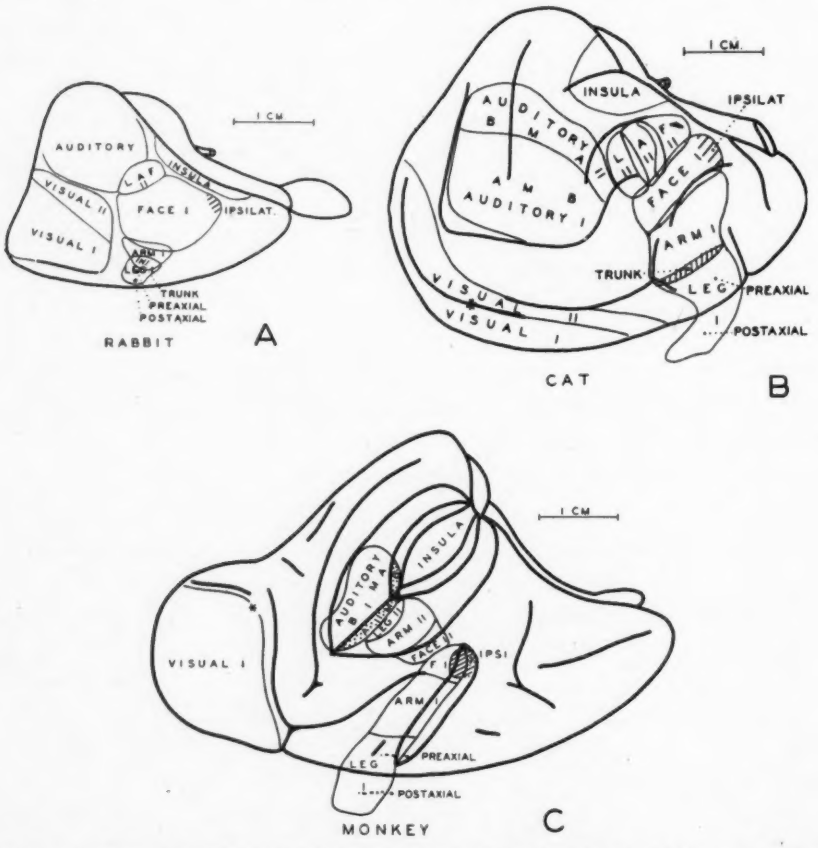

Fig. 6.—A, B, and C, These three figures show in the brains of rabbit, cat, and monkey the position and relative sizes of somatic areas I and II, of visual areas I and II, and of auditory areas I and II. Auditory area II is dotted in C. The second visual area has not yet been defined in monkey, nor have auditory areas I and II been established in the rabbit. These studies on which these diagrams are based are discussed in the text, q. v. Note the position in each brain of the ipsilateral face area. The question mark in this area of the monkey indicates that we believe the ipsilateral area may extend onto the anterior bank of the central sulcus. In the diagram of the monkey brain the central and the sylvian fissures are represented as having been spread apart to reveal the anterior and posterior banks of the central and the upper and lower banks of the sylvian. The insula also is exposed. The asterisks in the visual areas of cat and monkey indicate the cortical point for the center of gaze in each species.

The letters *A*, *M*, and *B* in the auditory areas indicate the regions of projection of the apical, middle, and basal turns of the cochlea. The arrangement in area II is the reverse of that in area I. Visual area II is related to visual area I, roughly as a mirror image. Compare with the arrangement of the somatic areas, especially in the monkey.

Somatic I arm area gave responses chiefly on stimulation of the distal segments of the limb. One point for the shoulder was found within the ansate sulcus.

When it was found that ipsilateral and contralateral face areas were present both in pig and in sheep, search was made for a homologous area in cat and in dog. It was located in both species rather well forward in the hemisphere, rostral to the contralateral face area. The area in these animals, although of much smaller dimensions, showed the same general relationships as in the ungulates (Fig. 6, B).

DISCUSSION

The electrical technique of recording potential changes evoked by cutaneous stimulation now has been applied in this laboratory to the study of localization in somatic afferent areas of the cerebral cortex in monkey,[37, 38, 54, 55, 57] cat,[30, 38, 54] dog,[54] rabbit,[60] sheep, and pig. From these and from papers by Adrian[1, 2, 4] as well as from studies on auditory[49, 59] and visual [45, 46, 48] systems certain generalizations concerning cortical organization are possible. We shall summarize the story as it has developed and emphasize the basic principles which have been revealed. The results, we believe, have important bearings on the problem of cerebral localization in man, especially since it is now feasible to extend this type of study to man himself.

In 1937 Marshall, Woolsey, and Bard[10, 11, 37, 57] made use of the method of "evoked cortical potentials" (as distinct from spontaneous brain rhythms and potential changes induced by local applications of strychnine) to obtain a map of the somesthetic area of the postcentral gyrus of the monkey. They employed discrete light tactile stimuli, usually mechanical displacement of a few hairs on the body surface, and observed on the synchronized sweep of a cathode ray tube the amplified electrical potential changes produced in the cortex. For each cortical point examined in the total area, they defined the extent of cutaneous surface capable of activating it. The methods employed yielded a more detailed picture of the pattern of localization within this region of the cortex than had yet been achieved by other methods including that of electrical stimulation of the cortex of conscious man.[25, 41]

The main facts already known for man were confirmed, but, in addition, certain new features were established, for example, the pre- and postaxial surfaces of the hindlimb were found to be represented, respectively, on the dorsal and mesial aspects of the hemisphere (Fig. 6, C). It proved possible to analyze the cortical map in terms of dermatomes[57, 58] and to show that the spinal segments from T_1 through the last caudal (Ca_4) are projected to the contralateral postcentral gyrus and paracentral lobule in the same serial order as that existing in the cord and with overlapping at the cortical level comparable in degree to that seen in the corresponding dermatomes. The serial order of the cervical segments is retained, but on projection to the cortex these segments are reversed en bloc. This reversal brings the cortical fields of the upper cervical segments in contiguity with the cortical fields of the upper thoracic segments, and places the cortical field of C_8 adjacent to that of the trigeminal nerve. The reversal accounts for the fact that hand and face areas join in the cortex and

it also offers a reason for the face, arm, and leg subdivisions revealed by local strychninization.[23] The analysis has since been confirmed for the hindlimb area of the monkey by Bard and Woolsey[12] through direct electrical stimulation of dorsal roots and by mapping the corresponding cortical areas of electrical response.

With the exception of a portion of the face area (see discussion of ipsilateral face area to follow) the relations of cortex to skin are entirely crossed in somatic area I in the monkey and in all other animals, even in the pig where a few apparently bilateral effects from the snout probably resulted from mechanical spread of stimulus across the midline.

The pattern of localization within the postcentral gyrus of monkey has also been studied by Adrian.[2] His results, although less detailed, agree with ours in so far as they may be compared.

Adrian[2] also made comparative studies on rabbit, cat, and dog. The basic relationships of face, arm, and leg subdivisions, as seen in these species, were similar to the plan of localization found in monkey, although the various portions of the area differed in degree of development in different species (compare our Figs. 1, 2, and 6). The fundamental similarity suggested that the analysis of the postcentral area of monkey in terms of dermatomes might also apply to other mammals.

To obtain further evidence for this view a more detailed map of the cat's postcentral homologue was obtained by one of us (C. N. W.) in collaboration with Hayes[30] by studying not only the dorsolateral aspect of the hemisphere but also the mesial surface. All features of the postcentral area of monkey were identifiable in cat without difficulty. Monkey and cat differed in respect to the relative development of various portions of somatic area I but not in the basic plan of organization. The pre- and postaxial representations of the hindlimb seen in the monkey (Fig. 6, C) were also found in the cat (Fig. 6, B). Even in the rabbit, where the hindlimb area is the smallest so far defined, there is differentiation within somatic area I of centers for pre- and postaxial surfaces of the hindlimb (Fig. 6, A). One, therefore, must conclude that the basic plan of organization in somatic area I is the same in these different species. In the ungulates the story of somatic area I is incomplete but from what is already known it can be said that the same fundamental story seems to hold. Compare, for example, the basic similarities of the face area (F_1) of pig (Fig. 5) and monkey[10, 11, 57] in the following respects: details within F_1; relation of F_1 to ipsilateral face area; relation of F_1 to arm I and to somatic II (Fig. 6, C).

The arm subdivision of somatic I gave good responses from the distal segments but very little from more proximal parts. The area certainly is small by comparison with the face area of the pig but, until more thorough exploration has been made of the cortex within the ansate sulcus and on the mesial aspect of the hemisphere, it cannot be said that only the distal portion of the forelimb sends impulses to the cortex. It is perhaps significant that the only point giving responses from proximal parts of the limb was one on the rostral bank of the ansate sulcus (Fig. 5).

Campbell's[18] cytoarchitectonic map of the pig does not lend support to the view that a hindlimb sensory area may lie on the mesial surface, since his somatic sensory area does not extend to the mesial surface. It does not, in fact, include the area from which we recorded forelimb responses. Campbell's map, however, is probably in error in several respects. Certainly the area which he labeled "parietal" appears grossly to correspond to the motor cortex of sheep[9] and goat.[19] Moreover, the area which he called "motor" is located within a region which in sheep has been called preparietal by Rose.[42] In contrast with Campbell's finding in the pig, Rose showed in the sheep that area postcentralis extends onto the mesial surface of the hemisphere in a position where one might expect from our studies to find a hindlimb sensory area.

In 1940 Adrian[1, 2] described a "second" somatic receiving area in the cortex of the cat, situated lateral and caudal to the face area. He reported that the "second" area receives impulses from the contralateral "claws and ventral side of the toes," with forepaw responses occurring anteriorly and hindpaw responses posteriorly within the area. Marshall, Woolsey, and Bard[38] also noted responses in the forepaw portion of this area but they failed to recognize it as an area distinct from the postcentral arm area. Adrian[2] searched for a "second" area in dog and monkey but did not find it in either and suggested that the "second" area was a specialization for claw-mechanisms in *Felidae*.

We have devoted considerable attention to this "second" somatic system, because in class experiments it has been possible not only to confirm the presence of the area in cats but also to demonstrate its existence in dogs. This led us to make a detailed study of the area and to look for it in other animals. To date it has been found in all species examined. In addition to cat[54] and dog,[54] it has been studied in monkey[54, 55] rabbit,[60] sheep, and pig.

Although our observations on somatic area II were made first on cat, then on dog and monkey, later on rabbit, and still later on sheep and pig, it was in the rabbit that the full story of somatic II was first obtained. Subsequently more complete data were secured for cat and monkey and then observations were made on sheep and pig.

In the rabbit (Fig. 6, A) it was first shown that somatic II receives impulses not only from limbs but also from face. The area in each hemisphere, or in one hemisphere after removal of the other, is activated by tactile stimulation of all parts of both sides of the body. Largest potential changes are evoked on stimulation of face, limb apices, and tail, small ones from proximal parts of limbs, and smallest from trunk. Contralateral responses are larger than ipsilateral ones, and deepening barbiturate anesthesia suppresses ipsilateral responses first, those of contralateral face last. In the rabbit, face, arm, and leg subdivisions nearly coincide, but the foci of maximal response for these three parts of the body are separated by about 1 mm. The face subdivision of somatic II is directly contiguous to face subdivision of somatic I. The two are readily distinguishable, however, by differences in latency of response and by the detailed localization in somatic I. In the rabbit also, it was quite easy to show by ablations that each of the two somatic receiving areas has its own pathway into the cortex.

The story is essentially the same for the cat (Fig. 6, B). The chief differences are the somewhat larger size of the area in cat and a correspondingly greater spatial differentiation within the area for different portions of the body. There is no overlap, for instance, between face and hindlimb subdivisions.

In the monkey the area is somewhat larger still (Fig. 6, C). Its arm and leg subdivisions lie on the superior bank of the sylvian fissure. The face subdivision lies in part within the sylvian fissure and in part on the inferior parietal gyrus adjacent to the face subdivision of somatic I. Again face I and face II are distinguishable by latency differences and by the detailed pattern of representation in area I. Somatic area II responses are obtainable in the monkey only under quite light anesthesia, therefore it has been difficult to work with complete satisfaction on this animal. In this respect, too, the contrast with somatic I is great. Probably because of the suppressing effect of the anesthetic agent on somatic II responses it has not been possible as yet to demonstrate representation for trunk and proximal parts of the limbs in monkey. At first[54] we obtained responses only from the contralateral limbs. In this our first results on monkey resembled those of Adrian on cats,[1, 2] the limited nature of which may also be ascribed to depression by the anesthetic.

In the sheep and the pig most of the characteristics of somatic II already described for other species also have been established. As in monkey, responses from proximal parts of limbs and from trunk were small or absent, but both sides of the body were found to be represented in each hemisphere. With respect to differentiation of face, arm, and leg subdivisions, pig and sheep appear to take an intermediate position between rabbit and cat, if one may judge by the degree of overlap between subdivisions.

The ipsilateral face area has been found in all the animals studied in this laboratory (rabbit, cat, dog, pig, sheep, and monkey). In all of them it receives impulses from the same parts of the body, that is, from the lips, the lower side of the face, the tongue, the buccal cavity, and perhaps the pharynx. In all species it lies adjacent to an ipsilateral motor area for the same portions of the body (rabbit,[16] cat,[27, 53, 56] sheep,[9] monkey.[56] An ipsilateral motor face area similarly situated has also been reported for the goat.[19] In the monkey the ipsilateral motor face area lies at the lower end of the central sulcus on the precentral gyrus. In our own experiments[56] this area in the monkey was strictly ipsilateral but merged anteriorly and medially with the contralateral motor face area through a transition zone giving bilateral contractions. It may be noted that bilateral motor responses for the lower face have been reported for primates by several workers.[24, 32, 33, 50]

It is of great interest that those portions of the body which animal experiments have shown to be represented in the ipsilateral sensory and motor face areas are the parts of the body which have most often shown bilateral relations to the cortex in electrical stimulation of the brain of man.[25, 41] Penfield and Boldrey[41] (p. 440) reported bilateral sensations from lips, tongue, and mouth and bilateral movements of these same parts. They comment: "This bilateral response is in such striking contrast to its complete absence in the extremities that it must indicate a small degree of true representation of bilateral function

in each hemisphere." From our own finding that the ipsilateral sensory area lies chiefly within the central sulcus in monkey and from the fact that much of the motor area in man is within the central sulcus, it seems likely that truly ipsilateral sensory and motor face areas in man may be largely buried on the walls of the central sulcus. The bilateral results described for man, then, would correspond to the results observed by us while recording from or stimulating the transition zones between contralateral and ipsilateral areas.

Dr. Dandy[20] has reported that ipsilateral sensory and motor functions in the domains of the trigeminal and facial nerves may be preserved in part after unilateral removal of one cerebral hemisphere in man. Possibly the ipsilateral areas just discussed have something to do with the preservation of these functions. One can scarcely account for the findings on the basis of somatic area II because arm and leg, as well as face, are bilaterally represented in this area.

What aspects of cutaneous sensory function are mediated by somatic area II is as yet unknown. A slowing of sensorimotor reactions (tactile placing) is the only impairment which has been observed to date following its unilateral removal in cats and monkeys.[12] We also know that somatic area II cannot compensate for loss of somatic area I in control of tactile-placing reactions.[12] Some recent interesting observations by Bender[13] on war injury cases, which he explains on the basis of bilateral cortical representation of the extremities, may prove related to somatic area II.

We have noted earlier in this paper that there are dual cortical areas not only for the somatic but also for the visual[45, 48] and the auditory[49, 59] systems (Fig. 6). Duality, therefore, appears to be a general principle of cortical organization. The position of somatic and auditory areas II between the rhinencephalon and insula on the one hand and the more highly differentiated areas I on the other suggest that the "second" areas may be phylogenetically more ancient and primitive. But the principle of dual representation is not limited to the cerebral cortex. This is clear from the study of Snider and Stowell[44] on afferent systems to the cerebellum. They found that tactile stimulation gave rise to electrical responses both in the anterior lobe and in the paramedian lobules. In the anterior lobe the responses are produced by ipsilateral stimulation only, while responses in one paramedian lobule can be evoked by stimulation of either side of the body, ipsilateral responses being larger than contralateral ones. These results suggest a parallelism between the anterior lobe and somatic area I, and between paramedian lobule and somatic area II. Indeed, Adrian[5] has shown that face, arm, and leg subdivisions of the rolandic cortex are related to face, arm, and leg subdivisions of the anterior lobe, and one of us (C. N. W.)[56] has found that somatic II and paramedian lobule are mutually interrelated.

One may deduce that undoubtedly separate pathways in the spinal cord serve these dual systems of the cerebral cortex and cerebellum. Possibly the systems concerned are those discussed by Brouwer.[17] If this is true then one must ask: Are there also dual systems of fibers in cutaneous, auditory, and optic nerves? It is tempting to think that this is so. We know that there are two types of auditory nerve fibers, the radial and the spiral. Do these respec-

tively project their messages to auditory areas I and II of the cerebral cortex? There are also the rods and the cones of the retina and two known types of vision, photopic and scotopic. Is photopic vision served by visual area I and scotopic vision by visual area II? (See Parsons[40] on dyscritic and epicritic visual systems.) Finally, one may recall that Head[31] postulated a dual system of cutaneous nerve fibers to account for phenomena associated with regeneration of peripheral nerves. It would be unwarranted to suggest a relationship between the dual cortical somatic systems and Head's postulated dual system of peripheral nerve fibers. Yet it is stimulating to think about this possibility, particularly since the detailed localization in somatic I and the less detailed localization in somatic II suggest that the function of the postcentral gyrus (epicritic?) would be much more seriously affected by confusion in the distribution of regenerated peripheral nerve fibers than would the function of somatic II (protopathic?). These are questions which one need not consider in a speculative manner only. It is quite possible, we think, to answer them by experiment.

SUMMARY

1. The somatic receiving areas of the cerebral cortex of pig and sheep have been studied by observing and recording the surface positive electrical potentials evoked in the cortex by mechanical stimulation of cutaneous receptors while the animals were anesthetized with pentobarbital sodium or with pentobarbital and chloralose.

2. As in other animals (rabbit,[60] cat,[1] dog,[54] and monkey[54]), two somatic afferent areas were found in each hemisphere: somatic area I, the homologue of the postcentral gyrus of primates and somatic area II, which in monkey lies on the upper bank of the sylvian fissure.

3. In both species an ipsilateral face area, receiving impulses from lips, side of face, tongue, palate and floor of mouth, also was found. This lies contiguous to and ahead of the contralateral face subdivision of somatic I. Whether it should be classified separately or as a part of somatic I has not been decided. Homologous areas have been found in cat, dog, rabbit, and monkey, and there is evidence that the ipsilateral face area may exist in man also.

4. In pig, face and arm subdivisions of somatic I were found; leg subdivision was not located but probably lies on the mesial surface of the hemisphere. In sheep, only the face subdivision of somatic I was studied.

5. Face, arm, and leg subdivisions of somatic II were demonstrated both in pig and in sheep.

6. In all animals so far studied the relations of cortex to skin are entirely crossed in somatic area I, with the exception of the ipsilateral face area. In the zone of transition from contralateral to ipsilateral face areas the relationships are bilateral.

7. Both sides of the body send impulses into somatic area II, but the contralateral responses are larger. Stimulations of snout, limb apices, and tail yield largest potentials. Few responses are produced by stimulation of trunk and proximal parts of the limbs, probably because the small responses to be expected (by analogy with cat and rabbit) are suppressed by the anesthetic.

8. Dual representation is not unique for the cortical somatic system. It has also been found in the visual[45, 48] and in the auditory areas[49, 59] of the cerebrum and in the tactile system of the cerebellum.[44] These dual systems provoke many interesting questions both for experimental and clinical investigation.

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THE SURGICAL TREATMENT OF VASCULAR ANOMALIES OF THE PREMOTOR AREA PRODUCING EPILEPSY

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AT THE 1935 meeting of the Harvey Cushing Society I reported on the treatment of three patients with jacksonian seizures due to vascular anomalies in the premotor area. In this paper were discussed certain peculiar findings in the ventriculograms of separation of the bodies of the ventricles by angiomas, and the marked temporary paralytic reaction following the coagulation of abnormal cortical vessels. The paper was withheld from publication because less than five years had elapsed following the operations.

That same year in a report on epilepsy due to vascular abnormalities of the brain, sixteen cases of vascular abnormalities in which operation had been done during the previous six years were discussed. Of these cases, one was due to saccular aneurysm coagulated successfully, another to an arteriovenous aneurysm treated by decompression,[1] another to vascular occlusion of the aqueduct of Sylvius,[2] six to vascular anomalies of the premotor area, and seven to angiomas or hemangiomas.

In the fall of 1936, a third report of six cases of epilepsy with vascular anomalies in the premotor region was read at the Pan-Pacific Surgical Congress.[3] Emphasis was laid on (1) signs of the premotor syndrome or the signs of a combined motor or premotor lesion in these patients, (2) the fact that ventriculograms in three cases revealed a new finding of slight separation of the body of the lateral ventricles just posterior to the anterior horns, which was found to be caused by deeply situated angiomas in the callosal region, (3) the observation that four of the patients had dural angiomas besides cortical vascular abnormalities, (4) the disturbance of cerebral vascular balance by the coagulation of the anomalous cortical and dural vessels and angiomas resulting in marked cerebral edema and transient hemiplegia that disappeared with the development of adequate collateral circulation, and (5) the fact that the repeated use of intravenous hypertonic solutions proved a more satisfactory procedure than decompression or repeated ventricular punctures in controlling this postoperative cerebral edema.

It is now timely to review these experiences in attempting to treat epilepsy due to vascular anomalies located in the premotor area and to present case reports of fifteen such patients operated upon from 1935 to 1941, with at least a five-year follow-up period.

Although the paper is limited purposely to lesions of the premotor region, the reader is referred to the 1928 monograph of Cushing and Bailey[4] on blood vessel tumors of the brain, to the 1928 article of Dandy[5] on venous abnormalities and angiomas of the brain, and to Dandy's[6] chapter on the brain in *Lewis' Practice of Surgery.*

The importance of the frontal lobes has been emphasized particularly by the experimental work from the physiologic laboratories at the Yale University School of Medicine, where careful observations of the effect of stimulation and destruction of small areas in this lobe had substantiated the observations recorded in a few scattered clinical reports.

Following the localization of forced grasping in Brodmann's cortical area 6 in monkeys by Richter and Hines[7] in 1932, a physiologic evaluation of this area was made by Fulton, Jacobsen, and Kennard,[8] who, besides securing the involuntary grasping reflex, observed spasticity and increase in tendon reflexes on the side opposite to the lesion. Later Fulton and Kennard,[9] and Kennard[10] determined that a lesion in area 6 caused the dermal temperature of the contralateral side to be lower than the normal side and produced transient edema and changes in the color of the skin and diminished the production of sweat.

These experimental observations on the premotor area were applied to the problem of a patient on whom a surgical removal of a cystic glioma restricted to the right premotor area was performed, and led to the enunciation of the syndrome of the premotor cortex in man,[11] consisting of impairment of skilled movements, forced grasping, spasticity, vasomotor disturbances, and increased tendon reflexes. The advances in the study of this region during the past ten years have been compiled in a recent book on the premotor cortex edited by Paul Bucy.[12]

Although certain authors had advised no interference in patients with anomalous cortical vessels, the first two of the patients operated upon in 1935 presented a problem that seemingly might be attacked by coagulation of the aberrant vessels. The procedures used in handling the stormy postoperative course of these two patients made possible a smoother treatment of subsequent patients with similar lesions, both at operation and in the postoperative period.

Reports of most of the following cases indicate that these epileptic patients had the syndrome of the premotor area alone or in combination with the adjacent motor area, and in them the anomalous vessels found at operation were coagulated.

CASE REPORTS

CASE 1.—Mr. M. C., aged 21 years, was seen in January, 1935, because of fainting spells of two years' duration.

On Thanksgiving Day, 1932, he was pulling out blasted tree stumps. That evening, while at a dance, he developed nausea and severe headache and was found next morning drowsy, delirious, and with a right hemiplegia. Within three weeks the hemiplegia showed improvement, but the right foot was never restored to normal, remaining cold and stiff with the toes in an extended position.

In the ensuing two years he experienced six attacks in which he fell unconscious, with the extremities rigid in tonic contracture.

On examination, the reflexes were increased on the right with positive Babinski and Rossolimo signs and a sustained ankle clonus. The right foot was weak, and the toes were spastic, cold, and in an extended position. Ventriculograms showed slight dilatation of the roof of the left ventricle (Fig. 1).

A diagnosis of cortical cicatrix following a subarachnoid and cortical hemorrhage in the left motor and premotor areas, due to some vascular anomaly, was ventured and exploration advised.

A left craniotomy was performed Jan. 15, 1935, under avertin and local anesthesia. A localized subarachnoid laking was seen. A cystic area could be palpated in the motor leg area (Fig. 2). This was tapped at a depth of 1 cm. and 10 c.c. of clear, straw-colored, non-coagulable fluid were withdrawn. When the needle was passed through the cyst, resistance by firm tissue was encountered for a distance of 3 cm. Three abnormal arteries, appearing on the mesial aspect of the hemisphere, entered the cortex of the premotor area and, on lateral retraction of the cerebrum, a large vein with branches from the leg and premotor area descended on the mesial aspect to enter a large abnormal venous channel in the falx several centimeters below the longitudinal sinus. This was coagulated, as was a moderately-sized artery from the dura which entered the premotor area. In an attempt to tap the anterior horn of the left ventricle, arterial blood was obtained without any sensation being experienced that a vessel wall had been entered, therefore it was suspected that an angioma had been punctured. The needle was not withdrawn until bleeding had stopped. The bone flap was replaced and closure effected.

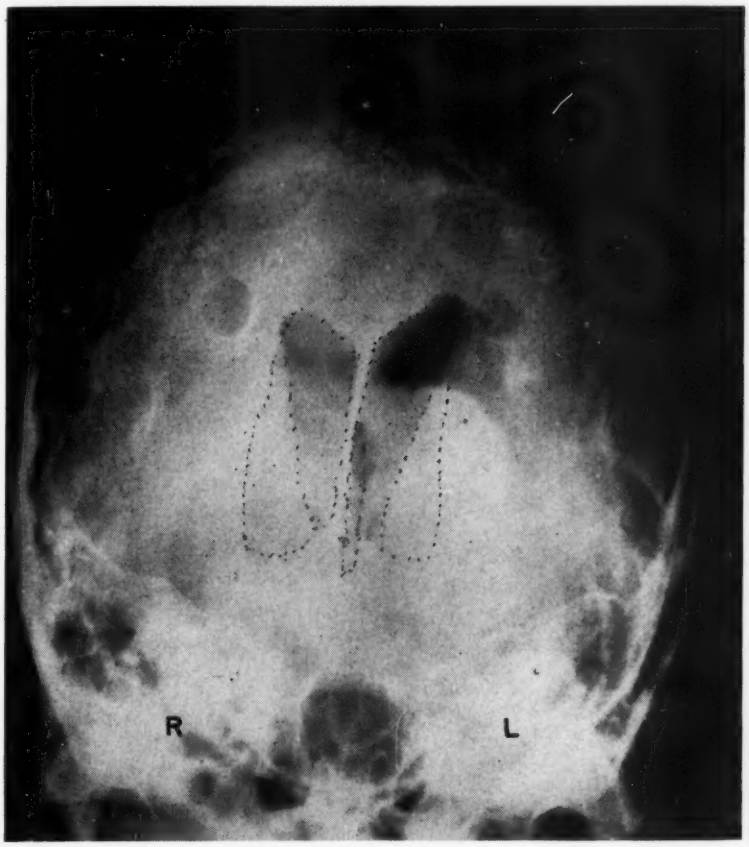

Fig. 1 (Case 1).—Anteroposterior view of ventriculogram showing slight dilatation of the roof of the left lateral ventricle from a subcortical scar.

On awakening from the anesthesia the patient had a slight aphasia and a right-sided weakness. A ventricular puncture gave 15 c.c. of clear fluid under moderate pressure.

During the course of the next two days the aphasia increased and the right paresis involved the hand more than the leg. Since it was feared that an extradural hemorrhage had developed, the bone flap was elevated under local anesthesia, January 17. A blood clot 1 cm. thick, which had formed beneath the bone flap, was removed. Because the dura felt tight,

although there was no subdural hygroma or hematoma, a subtemporal decompression was made. Subsequent ventricular punctures were planned, as well as a continuation of the limitation of fluid intake to 1 L. a day to control the edema.

Three right ventricular punctures, yielding 30 to 40 c.c. of clear fluid under moderate pressure each time, were made during the next day. No favorable response followed these taps and the patient became more drowsy, so on January 18 the bone flap was removed and preserved in alcohol for replacement later. Blood was again obtained at an attempt to tap the anterior horn of the left ventricle at this operation.

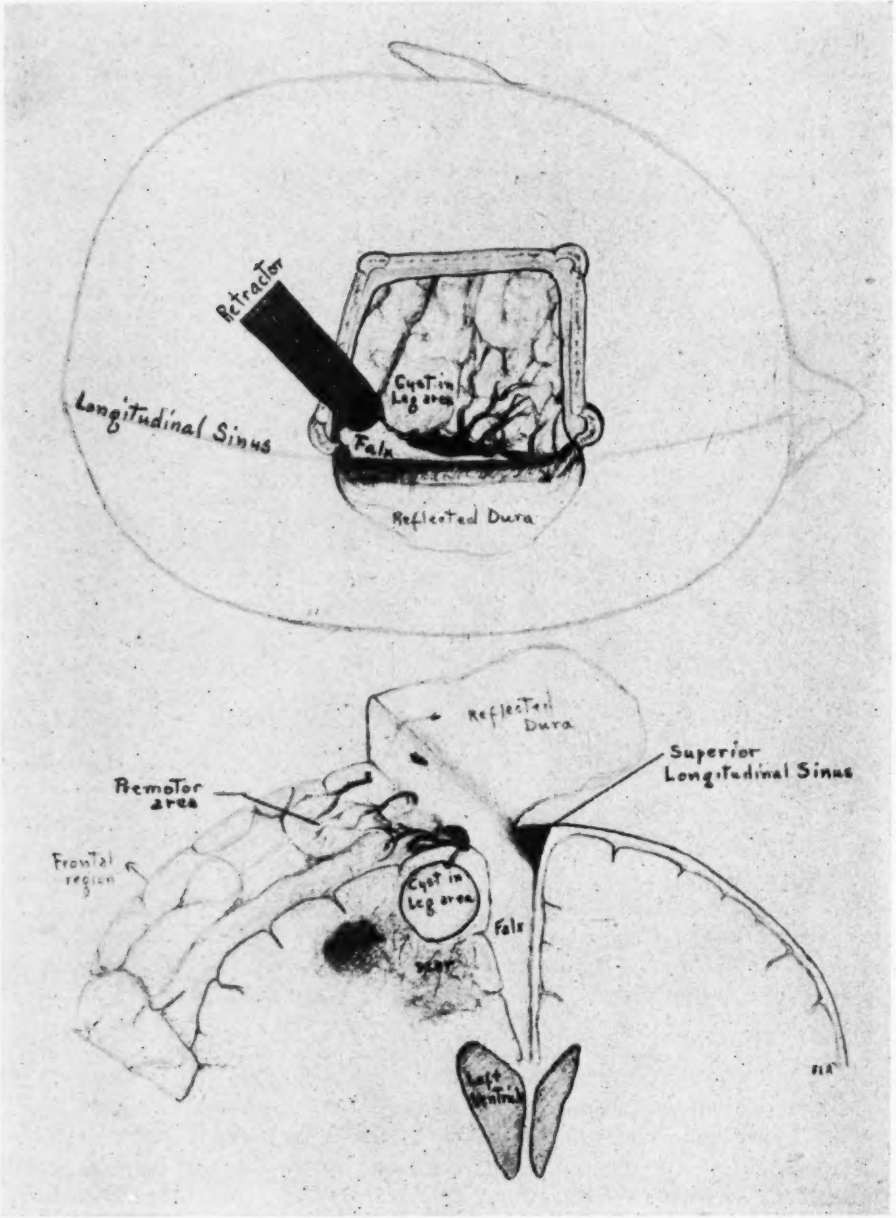

Fig. 2 (Case 1).—Operative exposure showing positions of cortical cyst, subcortical scarring, frontal angioma, and abnormal vascularity in left premotor region.

Further removal of right ventricular fluid by six punctures during the next day failed to make the patient more responsive, but after the intravenous administration of a hypertonic solution of glucose (12 per cent) and salt (5 per cent) a marked and favorable change was produced. The hypertonic solution was given twice daily for the next three days and, although a ventricular puncture was performed every six hours during the same period, it was felt that the deciding factor in his improvement followed the intravenous medications. By January 23 the decompression was soft, and rapid improvement occurred in the aphasia and right paresis.

An attack of influenza delayed his discharge from the hospital until February 9. At this time he showed a little hesitancy in speech with some weakness of the right upper extremity, and a more rapid recovery in the leg. The reflexes on the right were hyperactive with an ankle clonus and positive Babinski and Rossolimo signs. The spasticity in the right foot had disappeared and this limb was now as warm as the opposite one.

The patient returned on March 28, 1937, with a nearly normal gait, but a slight weakness and awkwardness still persisted in the right forearm and hand. Very little evidence of aphasia was found. The reflexes were still hyperactive but no pathologic reflexes could be obtained. The bone flap was replaced without any difficulty. The dura was under no tension. Only a few cubic centimeters of fluid were obtained in tapping the leg area. The cortex was not explored.

Since operation the patient has been examined on several occasions. The spasticity, coldness, and abnormal neurologic signs had disappeared, but he continued to have several epileptic seizures, clonic in nature, for three years, then occasional seizures in the next four years, with no seizures during the past three years. His speech was not rapid and the awkward, cold right upper extremity still was not much use to him as a grocery clerk.

Comment.—The onset of this lad's illness dated from Thanksgiving, 1932, when a subcortical hemorrhage, presumably from an angioma, occurred in the left premotor and motor areas. The cyst in the motor leg area, the subcortical cicatrix and angioma, revealed by operation, added support to this hypothesis. Abnormal arteries and veins in the premotor area, also disclosed by operation, were coagulated. An extensive cerebral edema developed postoperatively which was not affected by decompression, dehydration, and withdrawal of ventricular fluid, but was immediately and favorably influenced by the administration of hypertonic solutions given intravenously. Eleven years after operation found him with an awkward, cold, weak right upper extremity, slight evidence of motor aphasia when excited, and no seizures during the last three years.

CASE 2.—Miss L. P., a student, aged 25 years, was admitted in January, 1935, because of headaches, vomiting, and spasticity of the right hand for the previous ten days.

She had been subject to attacks of fainting for the preceding two years. In the first spell a friend noticed that the right hand and arm were spastically flexed. Subsequently she had transient periods of lightheadedness and headaches. By the fall of 1934 the headaches, usually occurring in the afternoon and found not to be due to eyestrain, were more frequent and severe, necessitating aspirin and barbital medication; the vertigo associated with nausea made her wonder if she had a brain tumor.

The recent episode came rather suddenly with nausea, vomiting, severe frontal headache, and spasticity of the right hand. She did not use the right hand because it was clumsy—she could not write with it. She complained of being dizzy, although objects did not move, and of having difficulty in walking. During a physical examination she paled and fell unconscious with tonic contraction of the right forearm and hand.

During the examination the patient was reticent in giving the history and kept her right hand under the covers. She was very despondent, with suicidal thoughts. Neurologically, hyperactive reflexes were found on the right side but there was no muscular weakness or

sensory change. The right hand was clinched but could be relaxed completely at times. Stroking the palm or pulling gently on the flexed fingers induced a marked spastic response. Her gait was unsteady and hesitant as the right leg was handled awkwardly and made her lurch to the right. Station was good, however. Touching the examiner's hand with her right foot or fingers was performed with hesitancy and inaccuracy. The right side of the body, especially the fingers and toes, at times felt definitely cooler than the corresponding part on the left side. The Babinski, Oppenheim, Hoffmann, and Rossolimo signs were negative. The eye grounds showed a slight haziness of the right nasal disc. The audiometer reading was normal. Blood pressure averaged 150/80, although two years before it was recorded at 120 systolic.

Fig. 3 (Case 2).—Anteroposterior view of ventriculogram showing slight separation of bodies of lateral ventricles.

The tentative diagnosis of labyrinthitis was changed to either a left frontal tumor or vascular anomaly, and ventriculography advised.

The ventricular fluid was under increased pressure but the ventriculograms excluded a tumor of the frontal lobe since the ventricles appeared normal in size. There was, however, a slight separation of the bodies of the ventricles just back of the anterior horns (Fig. 3). A revised diagnosis of vascular anomaly in the left premotor area was offered.

A left craniotomy was performed under avertin and local anesthesia on March 2, 1935. The exposed dura contained a cavernous angioma (Fig. 4) several centimeters in diameter

and 3 cm. from the midline. On reflexion of the dura, seven arteries and veins were seen to extend from the dural angioma directly into the premotor cortex. There was a small area of subarachnoid laking with arachnoid opacity in this region. The cortex felt normal. The abnormal vessels from dura to cortex were coagulated, then the dural angioma was coagulated and excised. Fascia from the temporal muscle was used to replace the excised dura, and then the bone flap was replaced.

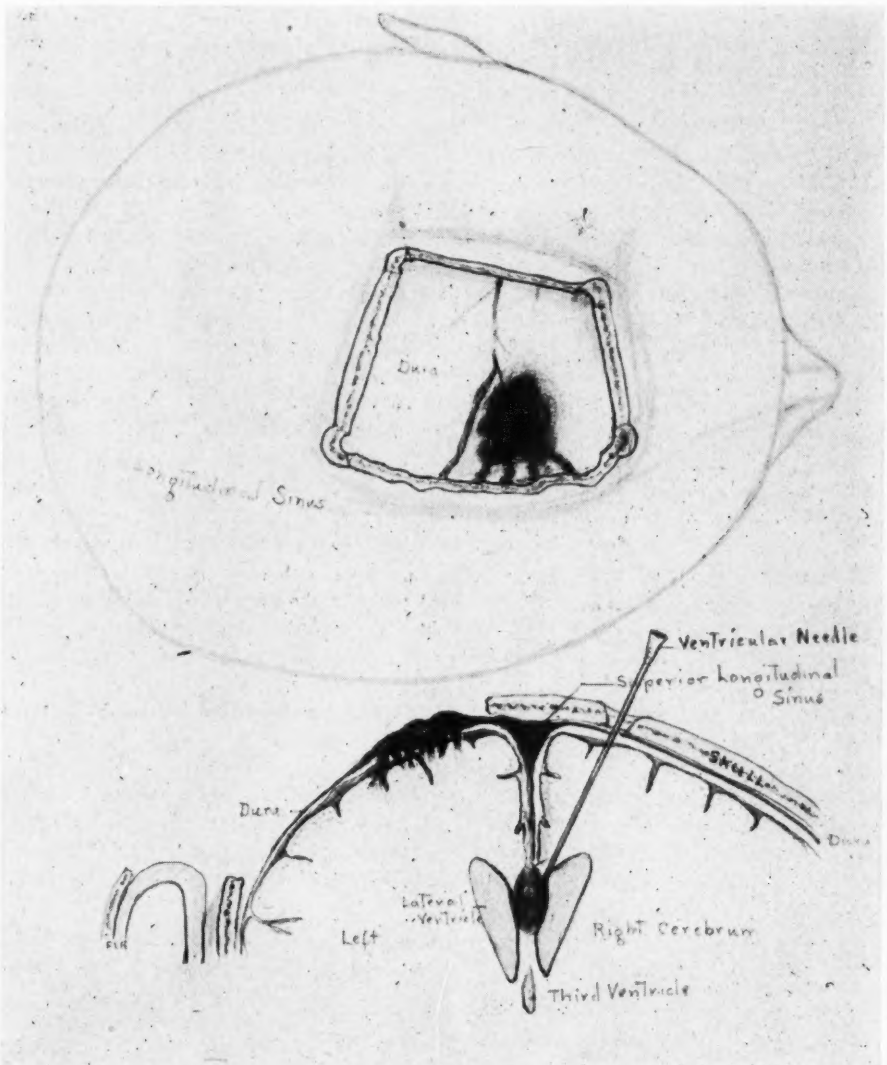

Fig. 4 (Case 2).—Operative exposure revealing the dural angioma with its seven arteries and veins entering the left premotor cortex, and the callosal angioma separating the lateral ventricles.

By evening the patient was awake, talkative, and without any right-sided weakness or spasticity, but the following day she was slightly drowsy. In view of the experience with the first case, ventricular puncture was done with the release of fluid under slight pressure. Intravenous injections of hypertonic solution of glucose and salt were given also, which made the patient more responsive. However, the second postoperative day found her drowsy and with

a definite right-sided paresis and aphasia which were not influenced by ventricular tap or hypertonic solutions to any marked extent, so a decompression was advised.

Before the bone flap was elevated, when an attempt was made to tap the right ventricle, through an anterior trephine opening, the needle encountered arterial blood without the operator feeling any resistance of a vessel wall. One ounce of blood flowed out briskly. By a slight shift of the needle, brain tissue came out which was pushed into the angioma by the stylet. This procedure caused a cessation of the bleeding and the needle was withdrawn. It was assumed that the needle's point had entered an angioma which had produced the slight separation of the lateral ventricles as shown by ventriculography. The bone flap was then elevated but only one-half ounce of clotted blood was found extradurally. As the dura was tight, a left subtemporal decompression was made.

The following two days showed an improvement in the patient's condition. Spinal instead of ventricular punctures were done, the fluid being slightly blood tinged, and hypertonic solutions were given intravenously twice daily. On March 6 the decompression was flat, the spinal fluid was straw colored, the blood pressure was around 125/75, and the patient was speaking a few words. The right hemiplegia persisted. The next day the left hand was spastic for short intervals, but this was never observed subsequently. The right hand also became spastic again, but relaxed the next day. Pain in the right arm and leg was a complaint, followed on March 10 by movements of the right toes. Rapid improvement in the aphasia and right hemiplegia persisted, but sitting up and walking was delayed until the fourth postoperative week because of annoying vasoconstriction when the patient was in the upright position, as shown by facial pallor and cold extremities, with the right hand becoming spastic. On reclining her color returned, followed by the warming of her hands and feet and relaxation of the right hand.

By the sixth week the aphasia had cleared, she could use her hand for eating and writing, and she walked with slight support. The reflexes were hyperactive on the right side, with an unsustained ankle clonus but negative Babinski, Oppenheim, and Rossolimo signs.

The vasomotor disturbance disappeared about the fourth week after operation, when the decompression was slightly sunken and she could perform the finer coordinating movements of the right hand, write legibly, and walk without support. Occasionally the right foot felt a little cooler than the left, but the hands showed no difference in temperature. The systole blood pressure stabilized around 120 since the first postoperative week.

After three months all signs and symptoms had subsided and an adequate collateral circulation apparently had developed.

About one year later, however, she experienced two fainting spells and then, rather suddenly, developed dizziness, nausea, awkward gait, a left sixth nerve palsy, and slightly elevated blood pressure. Conjugated deviation of the eyes to the left was noted a number of times. Leakage from the midline angioma was postulated and in the course of two months, without any particular treatment, the symptoms and signs disappeared and she returned to her classes.

In 1938, the patient married, the first child was born in 1940, and subsequent children in 1941, and 1943. Her neurologic examination has remained negative, and there have been no seizures since operation.

Comment.—In this young woman, signs and symptoms such as attacks of unconsciousness when the right hand was spastic, headaches, vertigo, nausea, and vomiting, with recent development of spasticity and forced grasping in the right hand which, at times, was cooler than the left, and hyperactive reflexes on the right side, gave the localization of a lesion to the left premotor area. The presence of a tumor was eliminated by the ventriculograms, although a slight separation of the lateral ventricles in the anteroposterior view was noted. Exploration revealed an angioma in the dura from which arteries and veins entered the premotor cortex. Another angioma in the callosal region was found

by needle puncture, which explained the separation of the ventricles seen in the ventriculograms, and its leakage one year later explained the bizarre bilateral symptoms.

Coagulation of the dural angioma and its abnormal vessels to the cortex produced an extensive left cerebral edema which caused drowsiness, aphasia, and hemiplegia that was not affected by withdrawal of cerebrospinal fluid or intravenous medication of hypertonic solutions. Following a subtemporal decompression, further withdrawal of spinal fluid, and particularly the administration of hypertonic solutions intravenously led to a rapid improvement. As collateral circulation developed, the signs of an irritative lesion in the premotor area gradually subsided. Eleven years later she showed no residual neurologic signs and has had no seizures since operation.

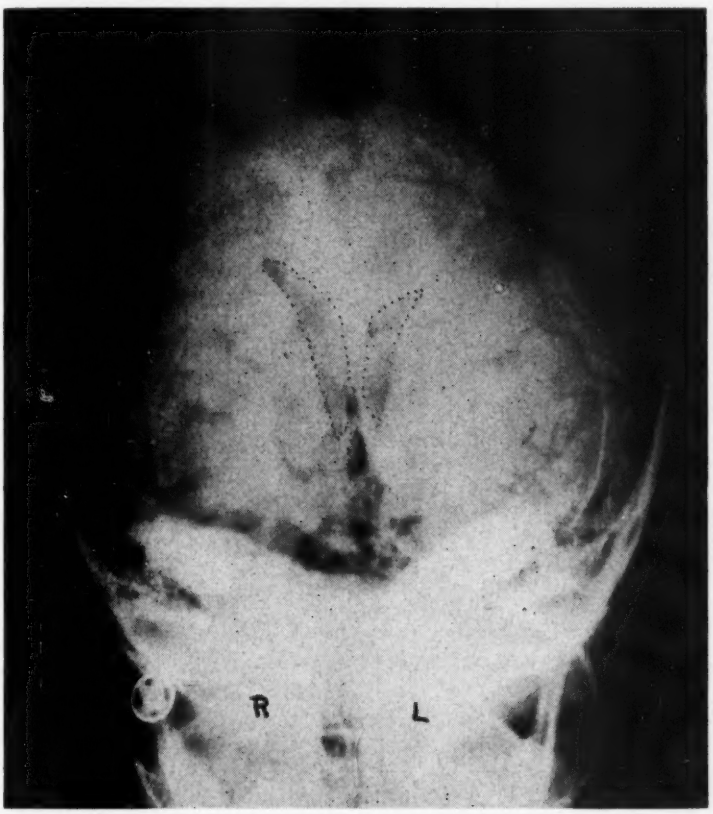

Fig. 5 (Case 3).—Anteroposterior view of the ventriculogram showing slight separation of the bodies of the lateral ventricles and slight dilatation of the roof of the right ventricle.

CASE 3.—J. E., male, aged 3½ years, was admitted April 15, 1935. One year previously he had spells of falling and becoming rigid. They were momentary and without loss of consciousness. If he were eating at the time he would drop the spoon and cup from his left hand. Since infancy he was inclined to use his left hand but was being trained to eat with his right hand. A slight stutter in his speech was noticed, with some hesitancy in his words. By November, 1934, he was having twenty to thirty attacks a day of falling forward, his face being flushed on arising, with dilated pupils. The seizures were more fre-

quently early in the morning. In February the physical examination was negative. He was placed on a ketogenic diet and ¼ gr. of phenobarbital twice daily which led to a marked diminution in the number of attacks and altered them to the extent that only nodding of the head was noticed. In March, however, the attacks of falling recurred and, on April 2, he had a convulsion in which he fell unconscious with clonic movements of the hands. His speech was indistinct for a short while after this seizure. Phenobarbital was increased to 1½ gr. daily. The roentgenograms of the skull and gastrointestinal tract were negative. No vestibular responses to caloric tests were found from the right vertical or horizontal canals, but there was vigorous response after douching the left ear.

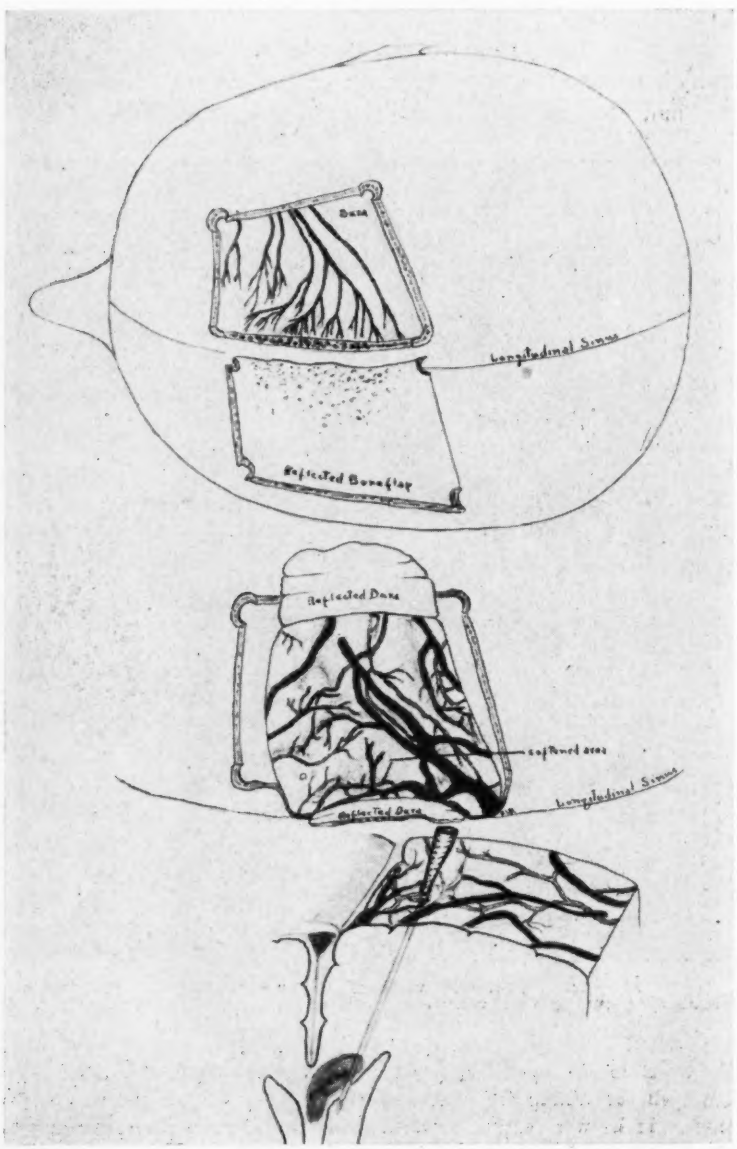

Fig. 6 (Case 3).—Operative exposure showing abnormal dural and cortical vascularity in right premotor region, the softened area, and the callosal angioma separating the lateral ventricles.

Examination on April 15 showed a very restless child with a tendency to stutter and to hesitate on simple words. He used both hands equally well. He sought his mother's hand in walking but when unsupported there was a tendency to throw out the left foot in a slow gait. While being examined his face flushed and he fell forward, striking his head against the crib, but he immediately arose crying and rubbing his head. The eye grounds showed nothing abnormal. The reflexes on the left side, particularly in the leg, were more active than on the right. Fanning of the left toes appeared on stroking the sole and Hoffmann's sign was positive on the left. The extremities had the same dermal temperature. A lesion, probably vascular, was suspected in the right premotor area and encephalograms advised.

Spinal air injection on April 16 showed small ventricles with bilateral absence of occipital horns. The subarachnoid channels did not deviate from normal. In the anteroposterior view the body of the right ventricle was a little larger than the left, particularly in the roof, and the lateral ventricles were separated just back of the anterior horns (Fig. 5), an appearance much like the ventriculograms in Case 2.

A diagnosis was made of vascular anomaly in the right premotor area, with a possible angioma in the callosal region separating the ventricles.

A right craniotomy on April 18 revealed the dura difficult to force from the inner table of the skull in the premotor region because of numerous branches of the middle meningeal artery entering the inner table. The bone was quite vascular along the longitudinal sinus. The abnormal, increased vascularity of the dura, however, did not extend into the cortex (Fig. 6). This portion of the dura was coagulated and destroyed. There was slight subarachnoid laking with a softened area palpable in the premotor region about 2 cm. from the midline. Just posterior to this area were rather large veins which formed a loose network close to the longitudinal sinus. Some of these veins were coagulated without causing any marked distention of the adjacent veins. A ventricular needle, introduced anterior to the softened area, was passed toward the ventricle. Blood slowly welled out, then, slightly deeper, cerebrospinal fluid was obtained. The needle was withdrawn until blood appeared and then the stylet was inserted in an attempt to put brain tissue into the angioma, which was situated between the lateral ventricles (Fig. 6). The bone flap was replaced and a right subtemporal decompression made.

The evening of the operation the patient's condition was good with no hesitancy in talking, or in the use of his left side, but the following day his speech was indistinct and hesitant. By the fourth day his speech was rapid and distinct. Slight twitchings of both hands were noticed at this time and the reflexes remained unchanged. A left jacksonian seizure occurred April 22. Suggestive athetoid movements of both hands, but more frequently on the left, were seen from the fourth to the seventh day. No spasticity and no forced grasping were noted.

In September, petit mal attacks became frequent with ataxia, increased reflexes on the right side, and bilateral Babinski and Chaddock signs. The decompression was full. An extension of the midline angioma seemed to explain the signs but, on repetition of the encephalograms, the same findings were obtained as before operation. Following this procedure the decompression became sunken and the spasticity and increased reflexes disappeared. The spells were less frequent, six occurring in three months, probably because he received 3½ gr. of phenobarbital daily. He accidentally took an overdose of barbital and died in June, 1937, a little more than two years after operation.

Comment.—The unusual type of seizures in this child of 3 years, occurring more frequently in the morning and associated with flushing of the face, at times with contractions of the left hand and with transient speech difficulty, together with the findings of increased reflexes on the left, fanning of the toes, and a positive Hoffmann sign as well as separation of the lateral ventricles and slight dilatation of the roof of the right ventricle shown in the encephalograms, suggested the diagnosis of a vascular anomaly of the right premotor area with

a probable angioma in the callosal region. This was confirmed at operation. A right subtemporal decompression seemed to make the convalescence smoother. However, transient aphasia, twitching of the fingers with athetoid movements, and a few left clonic seizures indicated that the cauterization of the dural vascularity and of several abnormal cortical veins, and the needling of the callosal angioma had disturbed the abnormal vascular balance. Some months later (as in Case 2) leakage from the callosal angioma gave signs of bilateral involvement.

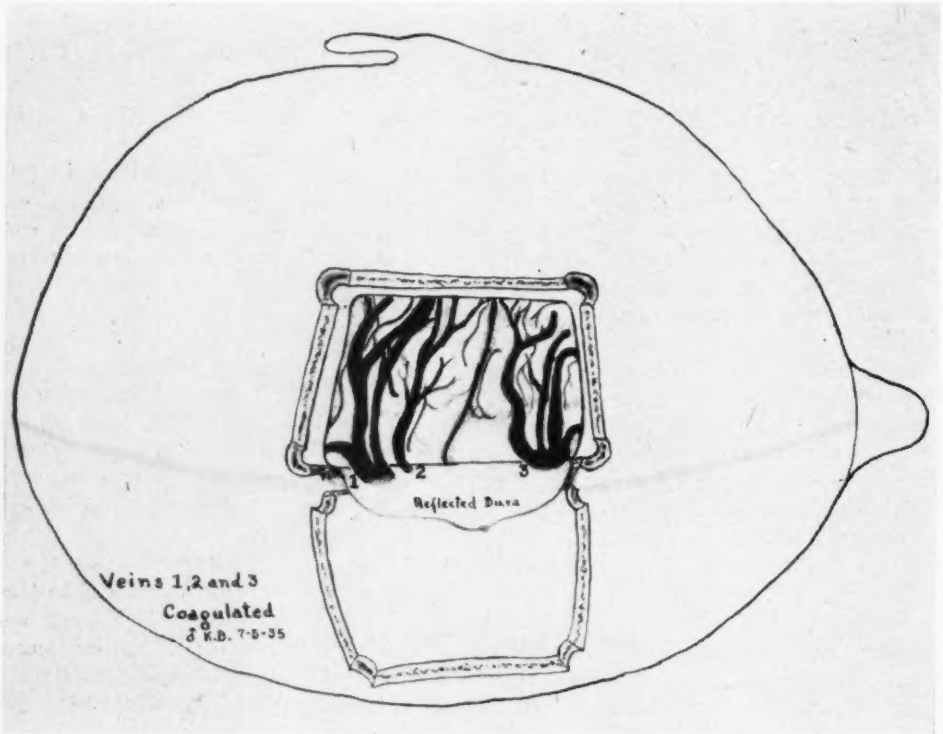

Fig. 7 (Case 4).—Operative exposure showing two groups (*1* and *3*) of large abnormal veins entering angiomas of the dura.

Case 4.—K. B., male, aged 13 years, was admitted in July, 1935. Two weeks after a severe blow on the head, in January, 1933, he had a nocturnal seizure beginning with a sensation of a knife thrust in the right foot, followed by tonic contractions of the right ankle and knee, spreading up the right lower limb. During the attack he was pale and his feet were extremely cold. Three weeks after the sixth attack, in September, 1934, increased reflexes on the right with a positive Babinski sign and fanning of the toes were found. When the patient was examined eight months later these signs had disappeared.

A left craniotomy for a probable vascular anomaly was performed on July 5, 1935. Slight subarachnoid laking was seen, and a softened area was felt in the leg region. Two groups of large abnormal veins situated on either side of a small rolandic vein were seen to enter angiomas in the dura (Fig. 7). These vessels and the dural angiomas were coagulated and a subtemporal decompression made.

A transient aphasia and a right hemiplegia disappeared by the tenth postoperative day. A report one year later stated that he was entirely well and markedly improved in dis-

position. In July, 1945, ten years after operation, his sister wrote that he was "fine," had never had a seizure since operation, and was serving overseas as a radio operator in the United States Army.

Comment.—This vascuar anomaly of the premotor cortex and dura, treated by coagulation, presented a fairly smooth convalescence probably because of the subtemporal decompression. No seizures occurred during the ten years since operation.

CASE 5.—E. W., male, aged 5 years, complained of dizziness and weakness since the age of 3 years. Shortly after the onset of the ataxic spells, he developed tonic flexor spasms of the right arm and wrist, as well as of the right face and leg. These spasms were likely to occur after playing or at night while he was sleeping. On examination the right side showed spasticity, forced grasping, and increased reflexes. Ventriculograms revealed slight separation of the bodies of the lateral ventricle, so that a diagnosis was made of a vascular anomaly in the left premotor area.

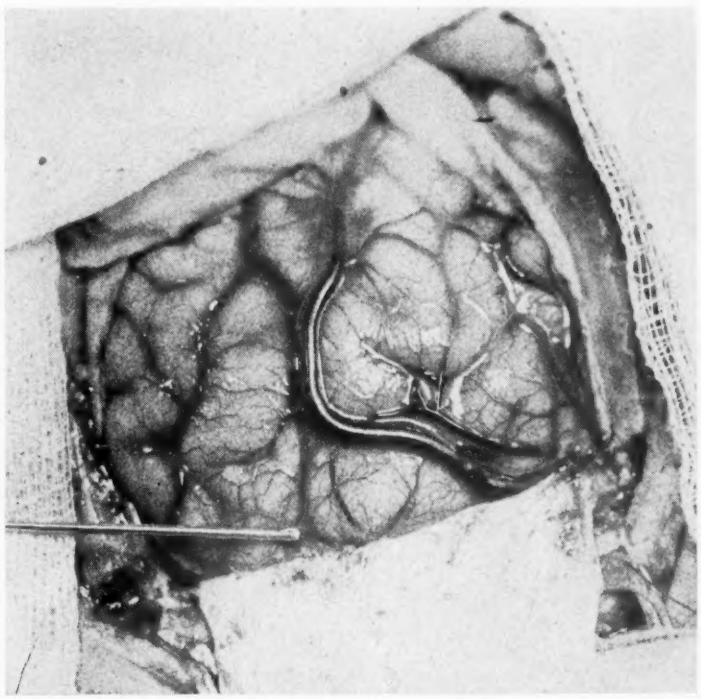

Fig. 8 (Case 5).—Photograph of exposed left premotor cortex, the pointer indicating where cortical vessels were entering a dural angioma.

A left craniotomy was performed on Aug. 20, 1935. Many vascular communications from the premotor area entered a large dural angioma (indicated by pointer in Fig. 8). These vessels and the angioma were coagulated. Histologic study of the dural angioma showed it to be composed of vascular channels filled with blood. A subtemporal decompression was made.

A right spastic paralysis had cleared by the tenth postopeartive day. Two weeks later tonic spasms recurred and still persisted two months after operation with the decompression remaining full. When examined April 5, 1937, one and one-half years after operation, he had had no grand mal attacks and had not taken any phenobarbital. The spasticity of the right arm had disappeared two months after operation but when he was excited the right side of

the face contracted slightly and at night a small contraction of the right fingers might occur. Neurologic examination was negative. The last letter received (in September, 1937) reported him well and gaining in weight. The family has since moved and no further follow-up could be obtained.

Comment.—The premotor syndrome in this lad was found to be caused by abnormal cortical vessels entering a dural angioma. Coagulation of these vessels produced a temporary hemiplegia, but a smooth convalescence probably resulted from a subtemporal decompression. The last report, two years after operation, stated that he was well.

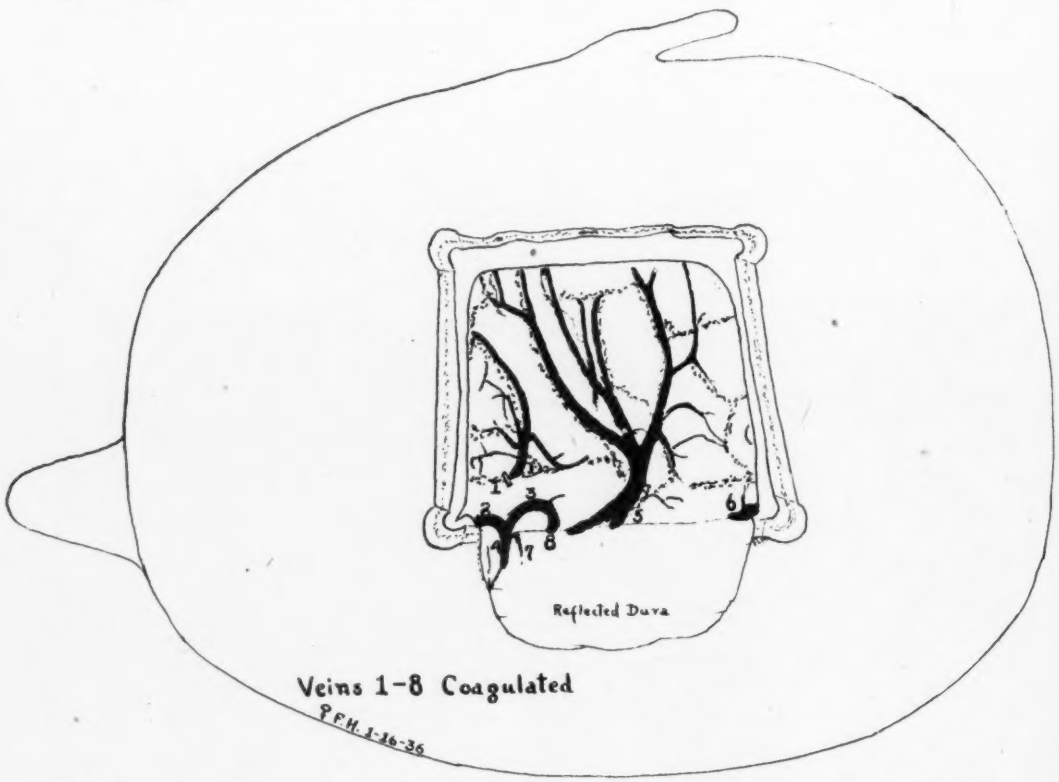

Fig. 9 (Case 6).—Operative exposure showing the many abnormal veins that were coagulated, including vein 5, that course beside the rolandic vein.

CASE 6.—Miss F. H., aged 24 years, was seen in May, 1931. An arrested hydrocephalus and a right torticollis had been present since childhood, possibly the effects of a difficult instrumental birth.

Illness at the time of admission dated from a fall in 1930, followed by weakness, heaviness, and awkwardness of the left arm and leg with, at times, dragging of the left foot. In 1931 the left hand and forearm were cold and spastic. Encephalograms showed a communicating hydrocephalus. In 1935 she dragged both feet, and both knees and legs were slightly spastic. Forced grasping in the left hand, and spasticity in the left wrist and elbow were demonstrated. Hyperactive reflexes, bilateral clonus, bilateral Babinski sign, and fanning of the toes were found. Both feet and the left hand were cold.

An irritative lesion in the right premotor area, probably a vascular anomaly extending over to the upper left premotor region, was diagnosed and a right craniotomy was performed on Jan. 16, 1936. No subarachnoid laking was seen. A large abnormal vein in front of area

6 ran from the cortex into the dura some 3 cm. from the longitudinal sinus. Silver clips were applied and the vein coagulated (Vein 1, Fig. 9). The rolandic vein was composed of three to five branches starting in the temporal region and uniting into a large trunk in the leg area (next to Vein 5, Fig. 9). In front of the ear two large tortuous veins entered the dura where they formed small angiomas. All the anomalous veins were coagulated, leaving only the rolandic vein. No subtemporal decompression was made.

The day after operation, spasticity was increased on the left side but with no increase in weakness. Spasticity and coldness were marked on the third and fourth days, but were gone by the ninth postoperative day. Five weeks later walking was improved, the limbs were warm, and spasticity and forced grasping were not demonstrated. Six months after operation pains developed in the left arm and elbow which became cold and spastic, with forced grasping and flexion contractures developing. In June, 1937, bilateral lumbar sympathetic block with alcohol warmed the lower limbs and slightly improved the spasticity of the lower extremities so that she could walk and climb stairs better. No further reports have been obtained from this patient.

Comment.—This girl had an arrested hydrocephalus from birth. Recent left-sided premotor symptoms and signs led to exploration and the coagulation of numerous abnormal veins, with only temporary relief.

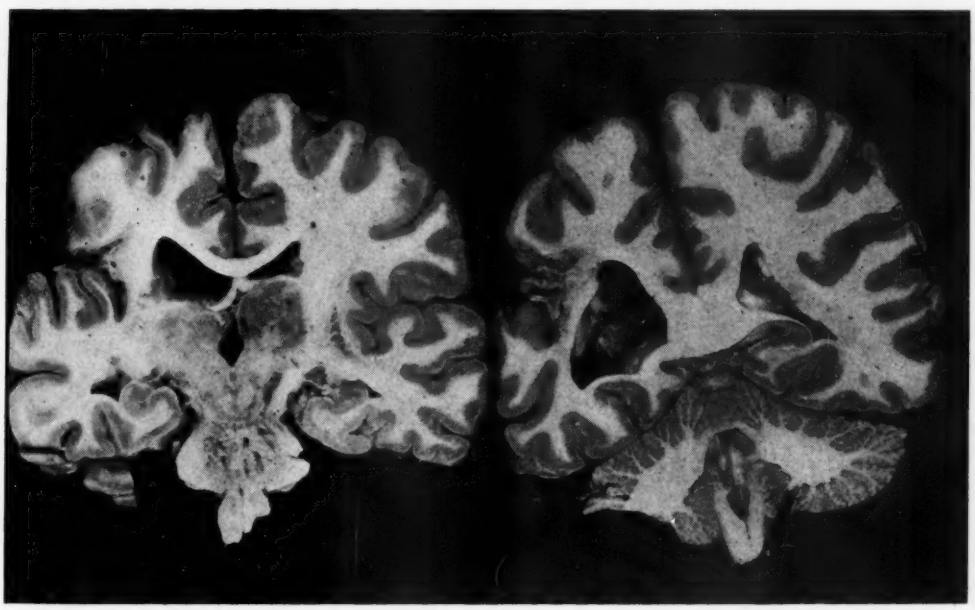

Fig. 10 (Case 7).—Photograph of brain showing the left cortical atrophy in premotor area and the compensatory dilatation of the left ventricle.

CASE 7.—Miss M. L. N., aged 23 years, was admitted Dec. 7, 1936, because of convulsive seizures of two years' duration.

A congenitally deformed right foot prevented walking until the age of 4 years. When 10 years old she struck her head on a rock. On examination five months after the onset of seizures a diagnosis was made of Little's disease and jacksonian epilepsy, and treatment started of phenobarbital and a ketogenic diet.

The seizures began in April, 1934, with the right arm suddenly flexed, and with spasms of the right face spreading to the right leg. Occasionally the clonic seizures became generalized. The attacks were without aura and in some seizures she lost consciousness.

On examination the right foot was arched and everted, and the right leg was small and weak. The right arm was weak, with deformity of the fingers. The reflexes were hyperactive on the right side with a questionably positive Babinski sign.

Because of the jacksonian seizures a left craniotomy was made on Dec. 12, 1936, at which time a cortical cyst was found in the parietal region. Anterior to the cyst the dura was adherent to the motor cortex. When the dura was freed a venous angioma was uncovered which was located, by stimulation, in the premotor arm and face areas. Several abnormal veins and the angioma were coagulated.

Following operation no aphasia or weakness developed. In February, 1937, Jacksonian seizures recurred but they were reduced to one or two a year under phenobarbital medication of ½ gr. twice a day.

In August, 1940, she attempted suicide with an overdose of phenobarbital and in November, 1940, she developed a lung abscess and empyema, refused surgical drainage, and died Nov. 7, 1940.

An autopsy showed multiple lung abscesses with empyema and a marked, localized unilateral left cerebral atrophy with compensatory dilatation of the left ventricle. The premotor area of the left frontal lobe particularly was affected (Fig. 10). Sections in this region showed a narrow zone of gray matter and beneath this were broad areas of glial tissue with scattered phagocytes containing granules of brown pigment.

Comment.—A birth trauma produced changes in the right arm and leg. Jacksonian seizures developed later on the right side. A cortical cyst resulted apparently from the birth trauma, although adjacent to it a cortical venous angioma was found and coagulated. Seizures recurred after operation. Four years later, autopsy showed cortical atrophy in the left premotor area.

Case 8.—W. C., male, aged 10 years, was admitted to the clinic in April, 1937. In 1932, he had fallen a distance of eight feet, struck his head on a concrete sidewalk, and remained unconscious for several hours. He developed headaches, personality changes, and increasingly frequent and severe convulsions in which his eyes turned to the right, the right arm and leg drew up, and, finally, tonic contractions of all extremities developed. Immediately following a seizure the Babinski, Oppenheim, and Gordon signs were positive on the right side but, at other times, the neurologic examination was negative. Memory loss and a personality change indicated by disagreeable, impulsive, unreasonable, and cruel reactions made him a behavior problem. Encephalograms showed a small filling defect in the middle of the body of the left ventricle. Exploration seemed indicated because of an irritating lesion of the left motor cortex.

Operation was performed April 13, 1937. The premotor cortex had soft areas with many capillary vessels entering large cavernous veins that extended toward the longitudinal sinus. All of these large veins were coagulated, leaving only the rolandic vein. Recovery was rapid without hemiplegia, and he was dismissed April 21. On April 25 he fell from a swing, disrupting the craniotomy wound which became infected, later necessitating removal of the bone flap. He was entered in the State hospital for behavior disorders and convulsions, where he remained until January, 1938, when he returned home so improved that he entered high school. Only two seizures occurred from 1937 to 1940, under dilantin, 0.1 Gm. three times a day. However, in 1944 and in April, 1945, he was arrested for stealing. His seizures have been much less frequent than before operation.

Comment.—This lad's condition seemed to have originated from trauma, yet the large abnormal vessels, resembling a hemangioma, of the left premotor cortex did not appear to have resulted from scarring.

Case 9.—J. C., male, aged 10 years, was admitted to the clinic June 23, 1937, because of convulsions involving the left side of the face and upper extremity for two years.

This patient had been born two weeks prematurely and was a "blue baby" for several days.

Two and one-half years before entry he struck his head on a rock. Six months later the seizures started, usually at bedtime, with clonic movements of the left side of the face and neck occasionally progressing to the left arm. Encephalograms were normal. A diagnosis of a probable vascular anomaly was made.

Right craniotomy on June 26, 1937, revealed a small dural angioma over the upper part of the premotor area with cortical vessels entering it. These vessels and the angioma were coagulated. Palpation revealed a softened spot in the face area where a very large vein was also coagulated.

From the time of operation until the last report in March, 1941, he had had no seizures.

Comment.—Jacksonian seizures of the left side in this boy were caused by a vascular anomaly of the dura and cortex which was coagulated. He was normal with no convulsions during the four-year follow-up after operation.

CASE 10.—Mr. A. C., aged 20 years, was admitted Aug. 14, 1938, because of convulsions that began at the age of 13 years. These seizures started in the right hand, progressed up the forearm and arm, and then spread to the opposite arm. When the legs were involved in clonic contractions, consciousness was lost. Neurologic examination was negative except for sluggish mentality, with a slow speech.

A left craniotomy on Aug. 16, 1938, revealed numerous abnormal veins and arteries in the premotor area. On stimulation, these were found to be in the finger area. Just posterior to this vascular area a softened region, 2 cm. in diameter, was explored revealing a subcortical angioma, bluish in color, which blanched on pressure. A circumscribed coagulation gradually removed this angioma. The abnormal surface veins in the finger area were coagulated also. Pathologic study of the excised angioma showed it to be honeycombed by small branching endothelial-lined channels which were filled with blood, indicating a plexiform hemangioma.

Subsequent to operation, attacks without loss of consciousness occurred about once a month. During the war he worked as a stock clerk in a Naval Arsenal, the seizures being fairly well controlled by phenobarbital and dilantin. However, on the night of Sept. 1, 1945, he was found dead in bed, probably from a convulsion.

Comment.—Apparently, this patient's jacksonian seizures were caused by a cortical hemangioma in the premotor area. This was excised by coagulation. The convulsions after operation probably were initiated in the operative scar.

CASE 11.—D. C., male, aged 12 years, entered the hospital in September, 1938. His mother had had generalized convulsions for twenty-five years, since the age of nineteen years. The patient's seizures began four months before entry, with an aura of numbness in the left forearm, followed by clonic movements of the left upper extremity. Neurologic examination was negative except for a positive Babinski sign on the left side. Encephalograms, September 9, showed normal ventricles and subarachnoid channels, although roentgenograms indicated a rarefied region of the skull in the right hand area. A vascular anomaly was considered and exploration advised.

Right craniotomy, Sept. 12, 1938, exposed a thin and bulging dura beneath the rarefied region of the skull. When the dura was reflected the underlying cortex was raised irregularly with a few small cysts that were filled with either blood or dark brown material, suggesting a hemangiomatous tumor. Stimulation showed this to be in the hand area. This vascular tumor, 5 cm. in diameter, was excised by coagulation and the bed carefully fulgurated. Pathologic studies revealed a cavernous hemangioma with large thin-walled, endothelial-lined spaces filled with red blood cells.

Left-sided weakness with numbness developed after operation. By the end of October he could walk alone and could use his left hand. His reflexes were increased on the left side with a positive Hoffmann sign. The last examination, April 30, 1943, about five years

after operation, showed a weak left foot with unsustained ankle clonus, and an awkward but usable hand. He had had no convulsions since operation. Phenobarbital was discontinued two years after operation.

Comment.—This patient's jacksonian seizures were caused by a cortical hemangioma which was removed by coagulation. No convulsions have occurred since operation. He was left with a slightly weak foot in dorsiflexion and a clumsy but usable left hand.

CASE 12.—Miss M. D., aged 13 years, entered the hospital because of convulsions beginning at the age of 6 years. An aura of numbness and weakness in the left arm preceded the clonic seizures which started in the left upper extremity. By May, 1939, weakness in the left arm and leg had developed, gait was slightly ataxic, with positive Romberg sign and increased reflexes on the left side. Encephalograms in January, 1939, were normal. Electroencephalography in July, 1939, showed abnormal waves over the right motor area. Because of the long history of seizures, a preoperative diagnosis of possible vascular anomaly was made.

A right craniotomy on July 11, 1939, showed slight subarachnoid laking with a softened area which, on stimulation, was found to be the arm area. The large vein which entered this area was coagulated. Two centimeters above the arm area two large and several small veins entered the cortex from a small dural angioma. These veins and the angioma were coagulated.

Two weeks after operation the left hand was still weak. In September mild seizures, without clonus of the arm, recurred. In January, 1940, the seizures were frequent, and weakness and awkwardness of the left arm returned. Encephalograms made in March, 1940, showed the body of the right ventricle a little larger and higher than the left. By January, 1941, although still having convulsions, the patient had much better use of the left upper extremity.

In April, 1944, the left hand was used almost as well as the right. In the previous five months eight mild seizures occurred involving only the left upper extremity, and without loss of consciousness. She was married in 1944. It was reported in August, 1945, that she continued to have an occasional mild seizure while on a dosage of 1 gr. of phenobarbital three times a day.

Comment.—A vascular anomaly in the premotor area was suspected in this girl because of the long history of jacksonian seizures and the recent development of left-sided weakness. Abnormal cortical veins from a dural angioma were coagulated. Mild jacksonian seizures still persist, although the left-sided weakness has disappeared.

CASE 13.—Miss R. R., aged 18 years, was admitted July 10, 1939, because of jacksonian seizures beginning with the onset of menstruation at the age of 14 years. The eyes rolled upward and to the right, then clonic seizures started in the right side of the face and spread to the right upper extremity and then to the right lower extremity, and often became generalized. Except for two or three attacks at each menstrual period, she was a normal, intelligent girl whose neurologic examination was negative. Electroencephalography gave abnormal waves without localization.

Left craniotomy, July 12, 1939, revealed a slightly thickened dura and considerable subarachnoid laking. In the area, identified by stimulation, supplying the face a number of small vessels, mainly arteries, entered a depressed region in the cortex. These arteries and one large vein were cauterized by electrocoagulation transmitted through a small cotton pledget.

Convalescence was smooth, with only a suggestive weakness of the right side of the face.

In August, 1939, a new type of seizure occurred on the opposite side, starting in the left side of the face with the left side of the body becoming rigid, while the right side re-

mained flaccid. These new convulsions continued through 1940 and 1941. In October, 1941, she deliberately became pregnant to cure her condition and had no seizures during the two months before a spontaneous abortion.

She re-entered the hospital in March, 1942, and electroencephalography still showed diffuse delta activity. Encephalograms revealed poor filling of the body and anterior horn of the right ventricle, with this ventricle and the third ventricle pushed 3 mm. to the left of the midline, suggesting a space-occupying lesion in the right frontoparietal region.

A right craniotomy on March 11, 1942, exposed a normal appearing cortex. Four explorations with a ventricular needle were negative. A subtemporal decompression was made.

A slight left paresis was present for two weeks and then disappeared gradually. Left-sided convulsions occurred occasionally, but finally ceased in October, 1943, and have not recurred for two years. She worked in a defense plant until her recent marriage, and she has taken no phenobarbital since 1943.

Comment.—Exploration revealed the apparent cause of the jacksonian seizures. Coagulation of abnormal vessels in the left area of the face stopped these right-sided convulsions. Shortly after operation, jacksonian attacks of the left side developed, and this condition led to a negative exploration of the right cerebrum three years later. The left jacksonian seizures became infrequent and have not occurred during the two years since October, 1943.

CASE 14.—Miss E. H., aged 15 years, was seen in December, 1939, because of peculiar attacks of abdominal pain occurring usually at night. These attacks started after an otitis media at the age of 5 years and were described by her mother as causing the child to cry out that her abdomen hurt. She would try to reach the bathroom, but usually became confused and lost. When she returned to bed the epigastric cramp might continue and she would have chewing motions of the mouth, and chilly, shaking limbs. She always urinated following an attack. The attacks became worse in the fall of 1939, five to twenty occurring in a month. Some were in the daytime, with a hot sensation, especially in the neck region, awkwardness of the left hand, and then an aching in the epigastrium which became cramp-like. Before an attack she was likely to have a voracious appetite. Following an attack she became pale and cold, and frequently had headache and nausea. Recently, with the epigastric pain, large white spots appeared on the left leg and disappeared when the pain ceased.

In 1936 appendectomy was performed for the cramps, and a recent gastrointestinal series and gall bladder visualization proved normal. Phenobarbital and low fluid intake did not affect the attacks. The reflexes were increased on the left side, with awkwardness of the left hand and foot.

Encephalograms in March, 1940, showed slight compression of the lateral wall of the body of the right ventricle without any shifting of the ventricle.

A right craniotomy on June 3, 1940, revealed a cortex covered with abnormal arteries and veins. No sylvian artery was seen but there were many small arteries in this region. At the upper end of the sylvian fissure, four large veins coalesced into a huge vein as it approached the base of the brain. After a hiatus in the face area, seven veins from the cortex extended upward and forward to the longitudinal sinus (lower left in Fig. 11). Slight subarachnoid laking was noted, except in several areas where the brain felt soft. Since subcortical angiomas were suspected, two of these soft areas were explored through a small nasal speculum but no angiomas were encountered. A spider web of abnormal vessels, mainly veins, was coagulated directly in front of the hand area (center in Fig. 11), some 3 cm. anterior to the rolandic fissure.

Epigastric pain and occasional spells continued until December, 1940. Then for one year she was free of attacks. In December, 1941, epigastric aching, with white areas on the left leg, returned, but the awkwardness of the left arm and leg was gone. In 1943 she had

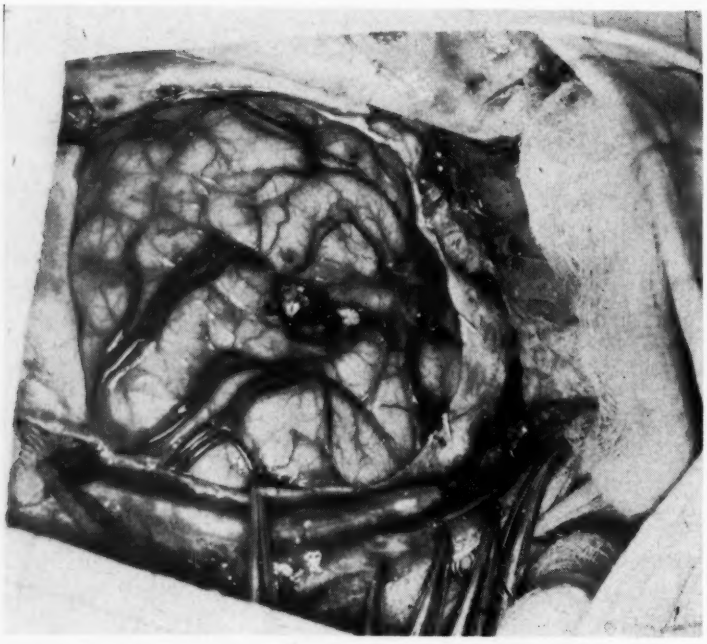

Fig. 11 (Case 14).—Photograph of the exposed right premotor region showing the spider web of abnormal vessels in the center of the photograph located in the hand area, and the two groups of veins coursing forward to the longitudinal sinus.

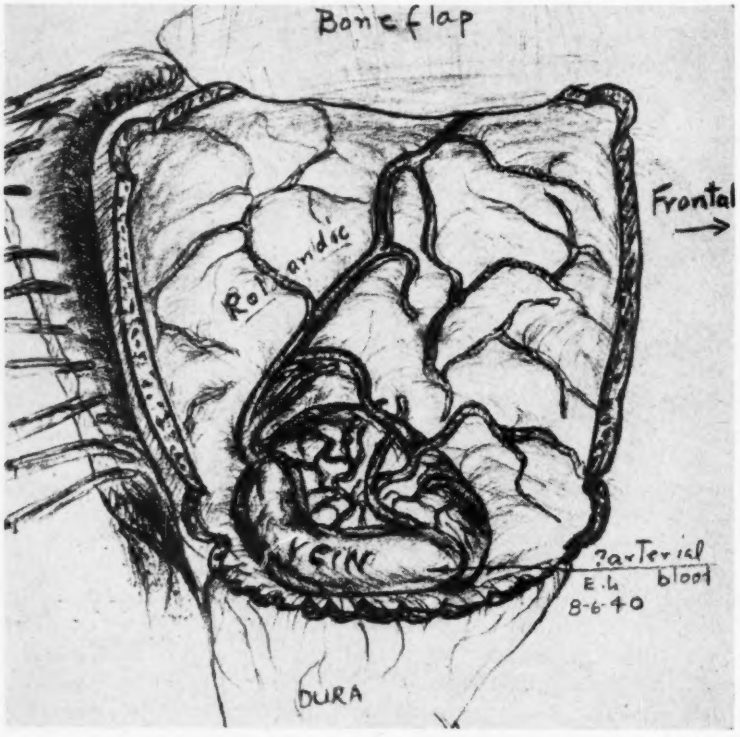

Fig. 12 (Case 15).—Operative exposure showing the large left cortical angioma that was coagulated two-thirds of the way around to stop pulsation.

eleven spells; in 1944, twenty-one spells; and in 1945, nineteen spells still characterized by crying out because of epigastric pain, "chilly" shaking of limbs, and urination. The white spots on the left leg have not appeared in the last two years.

Comment.—These attacks of epigastric cramps, with chewing motions of the mouth, and with white areas of skin appearing on the left leg, associated with increased reflexes on the left side and clumsiness of the left extremities, indicated a right premotor lesion. Abnormal cortical vascularity in front of the hand area was found and these vessels were coagulated. Improvement in the awkwardness of the left side and cessation of vasomotor blanching of the left leg followed operation. However, one or two mild spells a month of epigastric pain associated with shaking of limbs and urination occurred five and one-half years later.

CASE 15.—Mr. E. L., aged 24 years, was admitted in August, 1940, with a history of three jacksonian seizures during the previous two months. The attacks began in the right arm, where a numb feeling was followed by uncontrolled clonic flexions of the right forearm, which spread to the other extremities, and unconsciousness. The neurologic examination was negative. Roentgenograms showed the pineal body in normal position. Electroencephalography was reported normal but on review, after operation, prominent pulse waves were found in the center and to the left in the parietal region.

Left craniotomy on Aug. 6, 1940, exposed an unusual nest of veins in front of the rolandic fissure (Fig. 12). This pulsating angioma was 6 cm. in diameter and was composed of veins fully 1 cm. in diameter with numerous small arteries entering from the frontal region. This mass of vessels was tied or clipped and coagulated for two-thirds of the way around until pulsation could not be palpated. The destruction was carried inward for 2 cm. The angioma was not excised because it extended into the rolandic fissure and because of considerable bleeding. Fifteen hundred cubic centimeters of blood were transfused.

When the patient was dismissed, August 27, the right hemiplegia and motor aphasia were still present. By October, he could speak quite well and could move the right side, but could not walk well until the spring of 1941, at which time he could hold objects in the right fingers and hand. In September, 1941, when he returned to work in a service station, he had a slight limp and a spastic arm.

He was examined last in May, 1943, and there was no evidence of aphasia. The right arm and hand were usable but awkward, and were cool. The right leg was strong with a slight outward swing on walking. Phenobarbital, ½ gr. three times a day, has kept the seizures down to one to three a year.

Comment.—Although the large angioma which produced the jacksonian seizures was not removed completely by coagulation, apparently it has not recurred, and the occasional seizures were attributed to reaction to scar tissue.

SUMMARY

This report of fifteen cases of premotor vascular anomalies causing jacksonian epilepsy which were treated by coagulation of the vascular lesions during the six-year period, from 1935 to 1941, indicates that the operative procedure was feasible and satisfactory.

Nine of the fifteen patients had premotor signs.

Six of the fifteen patients, besides having the cortical vascular anomalies, had one or more dural angiomas.

Four of the fifteen patients had cortical angiomas or hemangiomas.

In three of the fifteen patients callosal angiomas were proved by ventricular needle puncture.

In these three callosal angiomas the ventriculograms showed a new sign of separation of the bodies of the ventricles.

Coagulation of the abnormal vessels frequently produced a transient hemiplegia, until normal collateral vascular channels developed.

Subtemporal decompression made a smoother convalescence if employed when there was evident disturbance of cortical circulation resulting from coagulation.

Intravenous hypertonic solutions aided materially in overcoming the cerebral edema that followed the disturbance of circulation incident to the operative coagulation.

Two of the fifteen patients had only temporary relief from seizures, after operation.

Seven of the fifteen patients had milder and less frequent attacks following operation.

Six of the fifteen patients, or 40 per cent, had no seizures after operation.

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THE IMPORTANCE OF AIR STUDIES IN A NEUROSURGICAL CLINIC

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THE introduction of ventriculography[1] and of encephalography[2] by Walter E. Dandy in 1918 and 1919, respectively, may be said truly to have inaugurated a new era in the investigation of diseases within the skull. It is, indeed, quite impossible to overemphasize the importance of these studies or to make anything like an accurate estimate of the lives they have saved or the discomforts they have relieved. If one considers air studies from the aspect of brain tumors alone, it is at once apparent how much this diagnostic measure has meant in the preservation of life. Before the era of ventriculography it was estimated variously that from 50 to 60 per cent of all brain tumors could be localized with sufficient accuracy to be disclosed at operation. By the use of air in the ventricles it has been possible to demonstrate the situation of practically all of the previously unlocalizable 40 to 50 per cent of intracranial growths. This means that by this single diagnostic measure nearly one-half of the total number of patients suffering from brain tumors are capable of having the tumors found at operation, and since we know that roughly 50 per cent of all intracranial neoplasms are capable of complete removal once they have been disclosed, the importance of air injections in brain tumor surgery is at once apparent.

It was perhaps quite natural that this type of investigation should have been taken up slowly by neurologic surgeons during the first few years after the announcement of the procedure had been made. This was due partly to the conservatism which most surgeons felt in regard to what might be the effect of air within the brain, but also to the fact that early experiences in some clinics had been followed by severe reactions and an occasional death. It was soon appreciated, however, that if the operation for removal of the tumor was undertaken immediately following the air injection, these difficulties did not result.* With this fact established, the value and importance of Dandy's discovery became manifest and with each succeeding year the employment of cerebral air studies increased enormously. The purpose of the present communication is to furnish an idea of the great usefulness of such studies in a neurosurgical clinic during the course of a single year.

At the Lahey Clinic from May 1, 1944, to April 30, 1945, a total of 429 cerebral air studies was carried out. Of these, 323 were encephalograms and 106 were ventriculograms. By ventriculography is meant the direct injection of air into the cerebral ventricles after making burr openings in the skull. Encephalography is the introduction of air (or oxygen) into the spinal subarachnoid space and thence to the cerebral subarachnoid spaces and ventricles by means of a

*In the present series of 429 patients subjected to air study there were 3 deaths. Two of these occurred following encephalography in defective children with marked internal hydrocephalus due to cerebral birth injury, and the other in a stuporous patient with brain tumor, following ventriculography before the immediately planned craniotomy could be well started. The total mortality, therefore, was 0.7 per cent.

lumbar puncture. In general, ventriculograms are preferred when there is evidence of increased intracranial pressure, whereas encephalography is used in conditions in which such pressure is not present.

USES OF VENTRICULOGRAPHY

The 106 ventriculograms were carried out largely on patients in whom a brain tumor or brain abscess was suspected. In 72 instances a tumor* was disclosed and in 3 the patients had abscesses. The remaining 31 patients had conditions such as increased intracranial pressure without tumor (arachnoiditis or encephalitis), cerebral vascular disease, intracerebral hemorrhage, and other infrequent lesions.

For the most part, ventriculography was performed in order to get information on the following three points:

1. *To Diagnose the Presence or Absence of a Brain Tumor in Patients Having Choked Disks and Other Pressure Symptoms But No Localizing Signs.*—Cases 1 and 2 are examples of the first point.

CASE 1.—E. La P., a 41-year-old woman, was admitted to the New England Deaconess Hospital on Aug. 30, 1944. At the time of admission she was entirely disoriented and uncooperative. Her chief complaint was "frontal headache" for an indefinite period, and her husband stated that during the past year she had become listless, neglectful of herself and her home, and her memory had become poor.

Neurologic examination showed memory loss and disorientation as noted, together with some drowsiness and inattention. There was slight weakness of the lower left side of the face, but otherwise the cranial nerves were normal, although the fundi showed some early edematous changes. She had slight questionable weakness of the left arm and leg and the deep reflexes were greater on the left. A lumbar puncture done elsewhere revealed a pressure of 260 mm. The chemical examination of the fluid was normal in all respects. Roentgenograms of the skull showed some displacement of the pineal shadow downward and to the left according to one examiner, but this could not be confirmed by measurement.

A ventriculogram was carried out on Sept. 6, 1944. This showed the third ventricle displaced to the left, while the right lateral ventricle was displaced upward and outward. On the same day a right frontal craniotomy was performed and a deep, subcortical astrocytoma was subtotally removed from the medial portion of the frontal lobe and from the lateral ventricle.

Although the patient went through the operation in good condition she succumbed the following day to sudden respiratory failure. Necropsy revealed a chronic mitral and tricuspid endocarditis.

Comment.—While a frontal lobe tumor might have been suspected in this woman, such a localization without air would have been entirely guesswork and even if the correct area had been explored, the tumor might never have been found.

One patient (Case 2) had pressure symptoms and signs with no localizing evidence, and without air studies it was impossible to tell whether or not a tumor was present.

*During the year in question there were 126 brain tumors verified histologically by operation. In 81 instances one or the other form of air study was used previous to operation (ventriculograms 72; encephalograms 9). Air studies, therefore, were felt to be necessary in 64.2 per cent of subsequently verified intracranial growths. If one excludes 16 pituitary adenomas and 4 acoustic neuromas since they can almost always be diagnosed without air, the figure rises to 76 per cent.

CASE 2.—J. V. T., a 40-year-old man, was admitted to the New England Baptist Hospital on Jan. 19, 1945. The chief complaints were headaches and dizziness. The former had become severe during the three months previous to admission, although they had been present for two years. Dizziness had been noted at intervals for one year.

Neurologically there were no positive findings other than bilateral papilledema of 2 to 3 diopters. A ventriculogram was performed on Jan. 22, 1945, and this disclosed normal-sized ventricles which were in no way displaced. For this reason a right subtemporal decompression was carried out in order to relieve the pressure symptoms and choked disks. He was discharged on Jan. 31, 1945. On Oct. 9, 1945, he reported for the most recent checkup examination. The decompression area was soft and the fundi were within normal limits. He had had only occasional slight headaches, and had resumed his former job several months previously.

Comment.—This patient represents a well-known class of individuals who have evidences of increased intracranial pressure and are suspected of having a brain tumor. The latter, however, can be ruled out only by demonstrating a normal ventricular system by the use of air. The underlying condition is probably an arachnoiditis or meningo-encephalitis.

2. *To Localize a Brain Tumor When Neurologic and Other Data Gave No Indication of Its Site.*—An excellent example of such a tumor is illustrated by Case 3.

CASE 3.—I. B., a 31-year-old married woman, was admitted to the New England Deaconess Hospital on May 4, 1944. She had complained of frontal headaches for nine months. These had been persistent until two weeks before admission when they had ceased. At times she had had mild nausea and periods of momentary blindness, but no other subjective symptoms.

Neurologic examination disclosed bilateral choked disks of 4 diopters with considerable new tissue formation embedding blood vessels over the disks. Venous tortuosity and engorgement were marked. There was questionable weakness of the lower left side of the face and, curiously enough, some question of sensory loss throughout the whole *right* side. Visual fields were normal.

On May 6, 1944, a ventriculogram showed displacement of both lateral and third ventricles to the left, with displacement backward of the right frontal horn as well. A right frontal craniotomy was performed the same day and an extensive soft glioma occupying the whole right frontal lobe and the anterior portion of the temporal lobe was radically but subtotally extirpated. Pathologically, the tumor proved to be a fibrillary astrocytoma.

The patient made a good recovery and was discharged on May 27, 1944. She was very well and resumed all her usual activities for sixteen months, when she again began to have intracranial symptoms. She was reoperated upon on Nov. 21, 1945, and the recurrent growth once more cleaned out. She has made a good recovery from this secondary operation.

3. *To Locate the Site of a Tumor More Exactly, Even Though Certain Localizing Evidences Were Present or When False Localizing Signs Happened to Exist.*—The third point is an extremely important function of a ventriculogram. Many patients with brain tumors show certain neurologic features which localize the growth to some extent, such as slight weakness or sensory changes on one side of the body, homonymous visual field defects, or possibly cerebellar signs which are not conclusive. In these patients a good-sized exploration over the suspected region might disclose the tumor in a fair number of instances, but by the injection of air into the ventricles the exact area for the craniotomy can be ascertained and the operation, therefore, undertaken with assurance. In modern neurosurgery a negative exploration should almost never occur.

Cases 4-6 may serve to illustrate the points referred to.

CASE 4.—R. A. F., a 30-year-old man, was admitted to the New England Deaconess Hospital on May 8, 1944. The chief complaint was headache which started in the frontal region and radiated backward, chiefly on the left side. In addition to headache, the patient had experienced dizzy spells, at which times he would see red and black spots before his eyes.

Neurologic examination revealed bilateral choked disks with an elevation between 1 and 2 diopters. The visual fields showed a right homonymous hemianopsia, the maculae being spared. There was likewise a slight hypesthesia of the right arm and leg as compared to the left, but no weakness of either side. There was a tendency to slight astereognosis for objects placed in the right hand. His deep reflexes were more active on the right than on the left.

A ventriculogram was performed on May 10, 1944, and showed both ventricles displaced toward the right and the posterior portion of the left lateral ventricle displaced forward, indicating a left occipital tumor. The growth was exposed by a left occipital craniotomy directly following the ventriculogram and a meningioma of the left occipital pole was completely removed. The patient made a satisfactory recovery and was discharged on May 26, 1944. He was seen for a checkup examination on June 19, 1945, at which time his right homonymous hemianopsia was unchanged, but otherwise he was in excellent condition.

Comment.—It was impossible to be sure from the neurologic and other clinical studies whether this patient's tumor was in the left temporal or the left occipital lobe. In the absence of aphasia, an occipital lesion might have been postulated, but the patient had given the information that he was partially right- and partially left-handed, so that the side of his speech center was somewhat in doubt, and therefore the temporal lobe could not be eliminated. In addition, ordinary x-rays of the skull had suggested a lesion near the optic chiasm due to erosion and sharpening of the left anterior clinoid process.

The ventriculogram clinched the situation and the bone flap could then be placed exactly over the lesion.

CASE 5.—T. S., was a 17-year-old boy who was admitted to the New England Deaconess Hospital on May 15, 1944. He had been well until six months previous to admission; since that time he had had increasingly severe headaches, and during the past month some unsteadiness in walking.

The neurologic findings were meager He had, at times, a slow nystagmus to the right and to the left, and his upward conjugate eye movements were slightly impaired, with a moderately quick nystagmus in this upward direction. He had bilateral choked disks of 4 diopters with beginning secondary atrophy. There was some swaying in the Romberg position and his gait was moderately unsteady. There was very little finger-to-nose ataxia. The deep reflexes were brisk and equal throughout.

Ventriculograms were carried out on May 17, 1944, and disclosed symmetrically dilated lateral ventricles as well as dilatation of the third ventricle. No air was seen in the aqueduct or fourth ventricle. On the same day a suboccipital craniotomy was performed, and a partly cystic, partly solid astrocytoma of the right cerebellar hemisphere was completely removed. He had slightly more than the usual temperature reaction following the operation, but otherwise made an excellent recovery and was discharged on June 10, 1944, greatly improved. A follow-up letter on Oct. 2, 1945, stated that he was very well and at work. He still had some slight unsteadiness in walking.

Comment.—In the days before ventriculography a cerebellar operation would doubtless have been carried out in this case and the lesion found, but the cerebellar symptomatology was far from outspoken and the demonstration of

dilated lateral and third ventricles left no doubt as to the tumor's location. In some patients with similar findings the growth might be located in the third ventricle or even in one of the frontal lobes, so that when there is any doubt at all an air study should always be instituted.

A good illustration of the use of ventriculographic evidence in the presence of what turned out to be false localizing signs is shown by Case 6.

CASE 6.—H. F. F., a 38-year-old woman, was admitted to the New England Deaconess Hospital on Aug. 17, 1944. She had a history of acute headaches and projectile vomiting, coming on some three weeks before admission. She had likewise had occasional dizziness and blurring of vision.

Neurologic examination disclosed slow nystagmus to the right and left and early choked disks of 1 to 2 diopters. The other cranial nerves were negative. Gait was somewhat unsteady and she swayed in the Romberg position but did not fall. Finger-to-nose tests showed slight ataxia, especially with the left hand. The deep reflexes were rather inactive and equal throughout.

On Aug. 21, 1944, a ventriculogram showed the lateral and third ventricles displaced to the left, and the right lateral ventricle was depressed in its posterior portion. A right parietal craniotomy was performed on the same day and a partly cystic but largely solid fibrillary astrocytoma was subtotally removed. The patient was discharged improved on Sept. 2, 1944.

Comment.—There is little doubt but that this patient would have had a cerebellar exploration if localization had been based solely on the neurologic findings, and of course no tumor would have been found. Ventriculography, therefore, was the only means of locating the growth.

USES OF ENCEPHALOGRAPHY

In patients who have no clinical evidence of increased intracranial pressure and in whom a brain tumor is not suspected we have used injections of air by the lumbar route for a wide variety of purposes. Occasionally an unsuspected intracranial new growth will be brought to light, and this was true in 9 instances among the 323 encephalograms performed during the period indicated. Perhaps one of the greatest diagnostic functions of encephalography is to distinguish between cerebral vascular disease and a possible tumor. Before the advent of air injections this problem arose in a large number of patients who were seen particularly on the medical wards in consultation. In a high percentage of individuals who came to necropsy a tumor was disclosed when the clinical diagnosis had been vascular disease. This error should almost never be made at the present time when the means of making a correct diagnosis are available by air insufflation.

Detailed case reports to illustrate all of the increasing uses of encephalograms are unnecessary. The following list comprises the majority of the problems for which we have used this form of air study:

1. To distinguish cerebral vascular and degenerative disease from tumor.
2. To demonstrate the presence or absence of a localized lesion in epilepsy.*

*Occasionally even in patients who have focal seizures air studies may appear normal in the presence of a brain tumor as shown by Poppen and Peacher,[3] but exploration in such cases is almost always indicated sooner or later.

3. To demonstrate the existence of an obstructive or a communicating hydrocephalus in children.

4. To demonstrate the presence or absence of a possible cerebellar or high cervical lesion (such as Arnold-Chiari syndrome) in patients in whom multiple sclerosis or cerebrospinal degenerative disease is the probable diagnosis.

5. To be sure that no possible intracranial surgical lesion exists in certain cases of presumed cerebral birth injury.

6. To use as a therapeutic measure in post-traumatic headache.

This latter function of encephalography deserves special mention. It was first utilized for this particular type of headache by Penfield[4] who, in 1927, reported on seven patients who had been relieved by this means. Penfield emphasized the importance of filling the subarachnoid spaces completely irrespective of whether the ventricles were filled. During the year which forms the basis for this report we utilized lumbar air insufflations on twenty-two patients as a therapeutic measure for headaches following trauma. At the moment, figures are not available as to the percentage of cures effected, but it is my strong impression that roughly three out of every four patients were relieved. It is certainly a most valuable aid in the treatment of this type of syndrome which seldom can be alleviated by any other means.

SUMMARY

The introduction of intracranial investigation by the injection of air into the cerebral ventricles and subarachnoid spaces was an epoch-making advance in neurologic diagnosis.

Examples of the value and extent of such studies in a neurosurgical clinic during the course of one year are summarized.

The employment of air injections has made possible the diagnosis and operable approach to a vast number of intracranial lesions which would otherwise have remained undisclosed and therefore inoperable.

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THE INNERVATION OF THE VEINS

Its Role in Pain, Venospasm, and Collateral Circulation

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RECENT investigations have shown that the nerves of veins, arteries, and lymphatic vessels involved in cancer are responsible for much of the pain.[1] For this reason a knowledge of the vascular innervation is a prerequisite for the treatment of pain in cancer by means of sympathetic operations. A review of the literature shows that the anatomic and physiologic studies to date have been chiefly concerned with the sympathetic innervation of the arteries, but the lymphatic vessels are intimately related to the wall of the blood vessels, and the sympathetic nerves of the veins are affected by the cancer.

In phlebitis and thrombophlebitis the investigations of Leriche and Kunlin,[2, 3] and Ochsner and DeBakey[4] have demonstrated that the sympathetic venous innervation plays an important role in the pain and in the vasomotor disturbances that accompany this disease. This shows the importance of determining the role of the innervation of the veins in pain, venospasm, and collateral circulation.

ANATOMICO-PHYSIOLOGIC FACTS CONCERNING THE INNERVATION OF THE VEINS

The few data available suggest, as Kuntz[5] has said, that the general plan of the distribution of the nerve components observed in the arteries obtains likewise in the veins. On the other hand, it is now admitted that the nerve supply to the blood vessels, and among them the veins, in addition to the efferent vasomotor components contains also afferent sensory pathways. The afferent fibers are components of the sensory cerebrospinal nerve roots but the extent of the distribution of such fibers and their relationship to other nerve components are not definitely known.

The large veins of the thorax and abdomen are supplied with nerve fibers that arise in the autonomic nerves located near them. The proximal segment of the large veins of the limbs near the sympathetic chain receives its nerve supply from the sympathetic ganglia and trunks located near them. But the peripheral vessels are supplied by sympathetic fibers which join them via the somatic nerves lying close to them (Kuntz[5] and Hirsch[6]). The sympathetic fibers running along the somatic nerves are distributed to the peripheral vessels at irregular intervals (Potts[7]).

From patients in whom periarterial sympathectomy has been performed, some observations led a number of authors to a different conception of the innervation of the vessels. It is their belief that long nerve fibers probably arise in the aortic plexus and its branches or in the sympathetic chain and, passing along the adventitia of the peripheral arteries, play an important role in the innervation of the proximal and peripheral parts of these blood

vessels. Other investigations, however, appear to indicate that the nerve fibers which innervate peripheral blood vessels are not derived from long plexuses from the splanchnic area (Kramer and Todd[8] and Hirsch[6]).

The anatomic investigations of Hirsch[6] on the distribution of the nervous elements in the larger vessels of the extremities demonstrated the nerve fibers running throughout the adventitia in larger or smaller bundles. Woolard[9] and others, however, claimed that the nerve fibers in the adventitia of the vessels are arranged in a continuous plexus which encircles the vessel.

Afferent fibers, according to Hinsey,[10] are present in the adventitia of the veins and the venules of the skeletal muscles. The afferent nerve fibers that penetrate the walls of the blood vessels terminate principally in the adventitia (Kuntz). Particularly in the larger veins, fiber terminations and end organs have been observed in the adventitia and are interpreted for the most part as receptive organs. According to Kuntz[5] some of these naked terminations consist of terminal branches or terminal loops and appear in the adventitia; other terminations are encapsulated end organs that, in the opinion of Hirsch,[6] are located only in the outer layer of the adventitia. There is no agreement as to whether, or not the nerve fibers also terminate in the intima of the blood vessels. Glaser[11] and others have maintained that nerve fibers also terminate in the intima, but the investigations of Hirsch[6] and Stöhr[12] have not confirmed this view.

As the volume of blood running through a blood vessel varies, in addition to other factors, with the caliber of the vessel, the vasomotor changes of the blood vessels induced by nervous control obviously play an important role in the circulation. It is now known that changes in the caliber of the veins are determined principally by the state of contraction of the inner circular and outer longitudinal layers of the musculature of these vessels (Kuntz). The active contraction of the circular muscles determined by impulses conducted through the sympathetic innervation of the vessels provokes vasoconstrictions. Inhibition of the circular muscles, increased pressure, or active vasodilatation increases the caliber of the vessels, but, as Kuntz[5] stated, the mechanism of the vasodilator fibers is not yet fully understood.

I have tried, in a number of patients, to add to the known facts and to approach the problem of the innervation of the veins by the physiologic and physiopathologic approach. I have analyzed (1) the afferent pathways of the innervation of the veins in relation to pain and (2) the efferent vasomotor pathways in relation to venospasm and also to the development of the collateral circulation of the veins.

PAIN AND INNERVATION OF THE VEINS

In several patients operated upon under local anesthesia I have made some observations that contribute to the knowledge of the mechanism of pain originating in the veins. In the femoral vein, mechanical or chemical stimulations of the adventitia induce a diffuse pain that can be relieved by anesthetizing the outer layer, just above the point of the stimulation. Anesthetic blocking of the

lumbar sympathetic chain also relieves the pain arising in the wall of the femoral vein.

On the other hand it is well known that a chemical drug, injected into a vein or into the adventitia, produces painful stimulations which differ in intensity. The outer layer of the veins (the adventitia) is particularly sensitive to painful stimulations. These observations are in agreement with the anatomic findings, for the adventitia is the layer of the vein in which, for the most part, the nerve endings and afferent fibers are located.

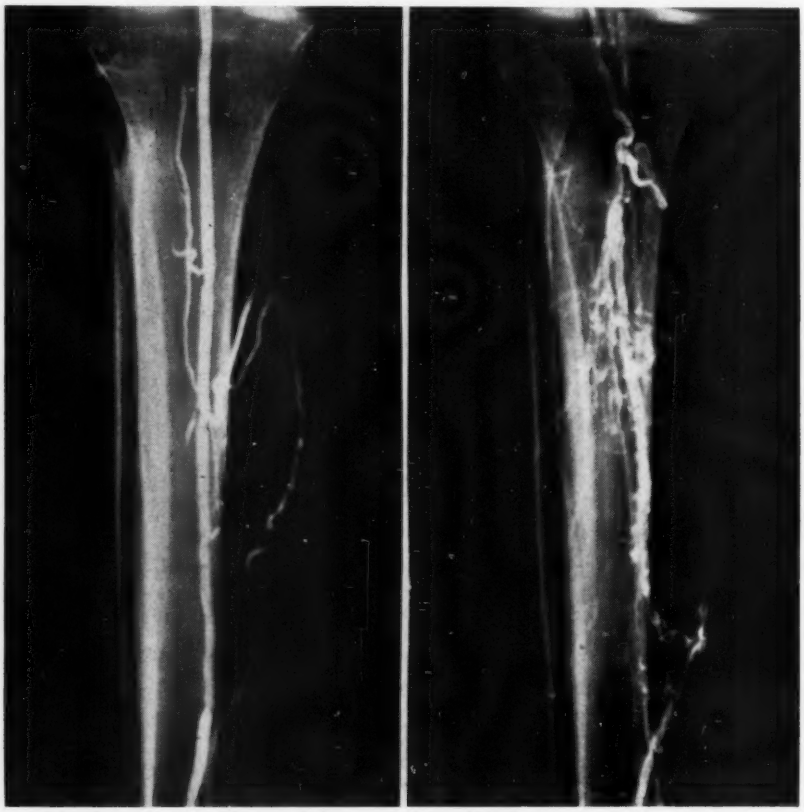
Penfield[13] found the dural sinuses to be sensitive to pressure, to traction, to heat and to electrical stimulation. The large cerebral veins that cross the subdural space to empty into dural sinuses produce exquisite pain when traction is exerted upon them. Bazett and McGlone[14] have observed that the pain experienced when a vein is punctured is of the same type as that produced when arteries are punctured. The sensation is that of a dull ache, not very painful, though difficult to endure and associated with reflex changes of an autonomic type.

The facts observed show that stimulation of the nerves in the veins may originate painful impulses. But it is necessary to demonstrate whether or not venospasm also plays a role in the mechanism of pain. Leriche[3] thought venospasm might be a cause of pain, and Ochsner and Debakey[4] have expressed a similar opinion.

In a series of fifteen patients with thrombophlebitis of the extremities, two of them bilateral, Ochsner and Debakey[4] performed the anesthetic block of the regional sympathetic ganglia with procaine. In these patients relief of pain appeared from fifteen to twenty minutes after the sympathetic block. In six cases the relief was permanent after a single injection, in eight cases after the second injection, and in the remaining cases after the third injection. In my own experience it was noted that the pain disappeared very quickly after the anesthetic blocks of the sympathetic chain, and for a longer period than the action of the anesthetic. This suggests that venospasm plays an important role in the mechanism of pain originating in the inflamed veins.

In two patients, one with acute phlebitis and the other with acute thrombophlebitis of the internal saphenous vein, I tried to determine the influence of the sympathetic innervation in pain and in venospasm, and the possible relation between these two elements. The first of these patients was observed four days after the beginning of acute phlebitis of the right saphenous vein at the level of the leg. Venographic study was made by the injection of 12 c.c. of uroselectan into the lower part of the saphenous vein. During the injection a compression equal to the minimal blood pressure of the patient was made with the armlet of an oscillometer, at the inferior third of the thigh. In the venographic picture of the lower half of the leg, the outline of the saphenous vein was only sketched, but in the upper half of the leg the vein was not permeable. By the same needle inserted into the saphenous vein I injected 15 c.c. of novocain (1 per cent). Forty-five minutes later a second venographic study was made, under the same technical conditions. The second venogram

showed the saphenous vein permeable all the length of the leg in the region where the clinical signs of phlebitis were evident. The pain disappeared after the novocain injection and the relief was maintained for a period of two hours. After this period the pain returned but it was not so intense as before; it was then completely relieved by a sympathetic block at the level of the third lumbar sympathetic ganglion. In this case the intravenous injection of novocain relieved the pain temporarily and at the same time relieved the spasm of the saphenous vein (Fig. 1).



A. *B.*

Fig. 1.—Acute phlebitis of the right internal saphenous vein in B.S.C., 60 years old. Venographies performed before (*A*), and forty-five minutes after the intravenous anesthesia (*B*), obtained by the injection of 15 c.c. of 1 per cent procaine hydrochloride into the lower extremity of the internal saphenous vein. In the second venography the venospasm shown at *1* in *A* disappeared and the saphenous vein is visualized in all its length through the leg (*2*) in *B*.

In a second patient with acute thrombophlebitis of the right saphenous vein, also studied by the venographic method, the saphenous vein was impermeable in all its length through the leg. With 30 c.c. of novocain (1 per cent) I blocked the third lumbar sympathetic ganglion of the right side, and two hours later a second venographic study was made under exactly the same technical conditions as the first. Twelve minutes after the sympathetic block the spontaneous pain had disappeared and the relief was maintained for a period of twenty-four hours. Then a slight painful sensation reappeared in the upper

segment of the saphenous vein; this was entirely relieved by a second anesthetic block of the sympathetic chain. Two hours after the first sympathetic block the patient was free from pain and the venographic study showed that the venospasm had disappeared and that the saphenous vein was permeable in the lower two-thirds of the leg. The upper third of the vein was obliterated. In this case there was also an increase in the development of the venous collateral circulation. The study of this case shows that the pain and the venospasm were relieved by the anesthetic block of the lumbar sympathetic chain. Moreover, the vasodilatation of the saphenous vein persisted after the anesthetic block of the sympathetic chain (Fig. 2).

A. *B.*

Fig. 2.—Acute thrombophlebitis of the superior third of the internal saphenous vein of the right leg in J.A., 44 years old. Venography was performed before and two hours after the anesthetic block of the third right lumbar ganglion. In the second venography it will be observed that the venospasm (*1,A*) has disappeared and the saphenous vein is permeable in the lower two-thirds of the leg (*2,B*). There is also an increase in the collateral venous circulation.

The results in the first patient show that anesthetization of the nerve endings in the venous wall by the intravenous injection caused the disappearance of the venospasm and relieved the pain in the vein. In the second patient interruption of the sympathetic pathways of the saphenous vein by anesthetizing the lumbar

chain was followed by relief of pain, more immediate and of longer duration. The venospasm also disappeared and the vasodilatation with increase in collateral circulation two hours after the sympathetic block persisted. These investigations demonstrate that the anesthesia of the nerve endings in the venous wall or of the afferent and efferent pathways in the lumbar sympathetic chain relieve pain and venospasm. And since the venous vasodilatation and the relief from pain lasted for a longer period than the action of the anesthetic, it seems logical to conclude that in these cases venospasm played a role in the mechanism of pain arising in the veins.

The pathway of pain fibers arising in the veins is indicated by the relief obtained by blocking the sympathetic chain; the afferent sensory fibers conducting painful impulses from the wall of the veins traverse the sympathetic chain to reach the spinal cord. That sensory fibers may pass through the sympathetic chain to join the somatic rami of the spinal nerves was claimed by Förster,[15] Kuntz and Farnsworth,[16] and others, but Burget and Livingston,[17] and Moore and Singleton[18] did not agree with this. My investigations show that interruption of the sympathetic chain relieves the pain in phlebitis and thrombophlebitis.

SYMPATHETIC INNERVATION OF THE VEINS AND VENOSPASM

A number of observatins support the view that the veins are innervated by vasomotor efferent fibers that control their tonus. De Takats,[19] discussing the role of venospasm in thrombophlebitis, frequently found a firm, hard cord, palpable in the groin during the first few hours of thrombophlebitis: this disappears under heat and elevation. He also referred to the contraction of the cubital vein induced by a traumatizing venous puncture, and the presence of spasm of the adjoining venous segments in patients with varicose veins treated by injection. I have also observed several times a venous spasm following the mechanical or chemical stimulation of the innervation of the wall of the femoral, saphenous, and cubital veins. It is interesting that the intensity of the venospasm induced by stimulating the adventitia of the veins varies in different individuals.

In 1938, Leriche,[3] suggested that phlebitis is dominated in its symptomatology and evolution by three factors: venospasm, the extension of coagulation, and that of the arteriospasm. The venospasm that he regarded as constant, and also as being the cause of pain, favored the extension of blood coagulation by decreasing the velocity of the blood circulation. Thus, an obliterated vein is not only functionally suppressed but it behaves in an abnormal manner through stimulation of the nerves in the adventitia. Leriche believed an obliterated vein behaves exactly like an obliterated artery. A thrombosed vein originates vasoconstrictor reflexes in contiguous veins and arteries. Ochsner and his co-workers[20] have demonstrated experimentally that localized chemical endophlebitis results in marked arteriolar vasospasm. They found the mechanism in these cases to be the result of sympathetic vasoconstrictor impulses that began in the involved venous segment. Ochsner concluded that these

reflexes are transmitted over the sympathetic system since sympathectomy or anesthetic sympathetic block prevents these reflexes.

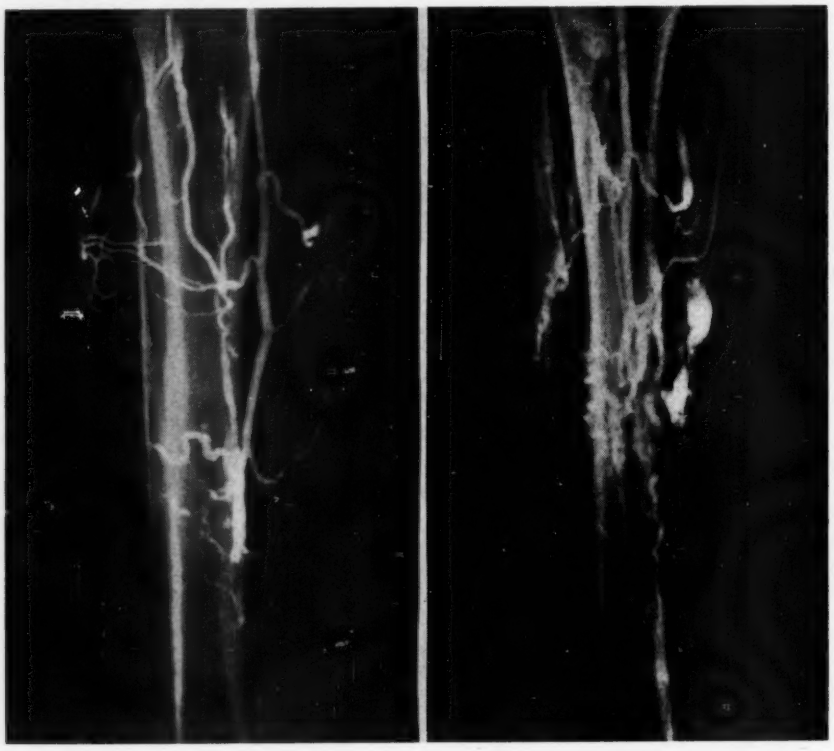
The investigations of Reichert[21] and Homans[22] indicate that in thrombophlebitis the venous obstruction is insufficient to produce the degree of edema that results from perivenous lymphatic obstruction. Zimmermann and De Takats,[23] however, concluded that the venous obstruction is the most important factor and that lymphatic obstruction plays a minor role in the mechanism of thrombophlebitic edema. From many experiments on the veins and lymphatics of dogs, Fontaine and I[24] have demonstrated that mechanical venous and lymphatic obstruction is of secondary importance. We have found that ligations, resections, or obliterations of the larger venous trunks and of their affluents, at the level of the root of a limb, cause only a transitory edema. We have observed also that when the lymphatics are obliterated by sclerosis in addition to the venous obstructions, the postoperative edema is of longer duration. Since the edema disappears completely in these experiments we are led to conclude, with Leriche, that neither the venous mechanical factor nor the lymphatic factor is enough to explain wholly the phlebitic edema. These facts and others that I have observed in clinical cases of phlebitis lead me to accept the view held by Leriche, Ochsner, and DeBakey that the innervation of the veins plays the most important role in the symptomatology and therapeutics of phlebitis.

In order to investigate the effects of venospasm, Papper and Imler,[25] made phlebographic studies of the veins of the lower extremity before and after anesthetic block of the lumbar sympathetic chain in three patients, the first with clinical chronic cellulitis of the right leg, the second with thromboangiitis obliterans, and the third with chronic recurrent migratory phlebitis of the right great saphenous vein. They confirmed the diagnosis of venospasm in these cases by blocking the lumbar sympathetics; following this the venous deformity produced by the narrowing of the spasmodic vein was abolished.

The investigations carried out by Dougherty and Homans[26] and Dos Santos[27] on the roentgenographic visualization of the venous system provided an interesting method of studying the role of the vasomotor innervation of the veins. In patients with acute phlebitis or with thrombophlebitis of the lower extremities, I have studied by this method the effects of interrupting the efferent vasomotor fibers on venospasm of the saphenous vein.

The existence of venospasm associated with acute phlebitis or with thrombophlebitis is clearly demonstrated in the two cases already mentioned. In the first patient, who had acute phlebitis of the right saphenous vein, there were clinical signs of phlebitis in all the length of this vein at the level of the leg. The venographic study showed that this vein presented a diminished caliber in the middle third and was completely impermeable in the upper third of the leg (Fig. 1). Forty-five minutes after the injection of 15 c.c. of novocain at 1 per cent into the lower part of the saphenous vein, the spasm had completely disappeared and the venographic study showed that it was uniformly patent throughout. The second patient had an acute thrombophlebitis of the right saphenous vein in the upper third of the leg. The venographic study

(Fig. 2) showed the vein to be totally occluded. Two hours after the third right sympathetic ganglion had been blocked with procaine hydrochloride, the venospasm had disappeared and the saphenous vein showed obliteration only in the upper third and permeability in the lower two-thirds of the leg. These cases demonstrate clearly that acute phlebitis or thrombophlebitis may be accompanied by venospasm and that this venospasm may be relieved by intravenous anesthesia of the inflamed vein or by the anesthetic blocking of the efferent vasomotor pathways in the sympathetic chain.



A. *B.*

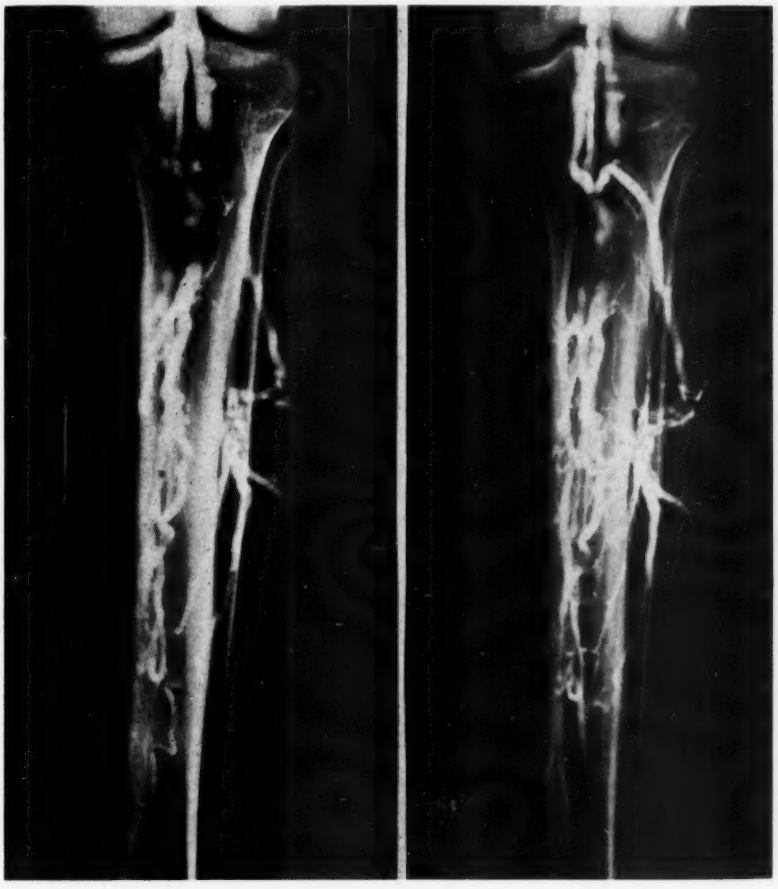
Fig. 3.—Thrombophlebitis of the deep veins of the right leg in A.L.C., 22 years old. Venography performed before (*A*) and one month later (*B*), after the treatment by eight anesthetic blocks of the sympathetic lumbar chain, (second, third, and fourth lumbar nerves). Following this treatment, pain and the edema disappeared and the later venography showed an increase in the development of the collateral venous circulation.

SYMPATHETIC INNERVATION OF THE VEINS AND COLLATERAL VENOUS CIRCULATION

Since the facts cited demonstrate the existence of a sympathetic efferent vasomotor innervation in the veins, it is important to know what part these nerves have to play in the development of collateral venous circulation.

Experimental and clinical investigations conducted by several authors (Leriche,[3] Linton and White,[28] Theis,[29] and others) have shown that interruption by sympathectomy of the vasomotor nerves of arteries increases the development of the arterial collateral circulation. My co-workers and I[30] have also verified these findings by experimental investigations on animals and have ar-

rived at the conclusion that the greater effect is obtained by section or by blocking the sympathetic ganglionic chain. But this conclusion is also exact so far as concerns the influence of the vasomotor sympathetic innervation on the development of the collateral circulation of the lymphatic vessels. We[31] have observed that if on dogs the femoral lymphatic vessels are interrupted by ligatures, anesthetic block or resection of the lumbar sympathetic chain increases the development of the collateral circulation of the lymphatic vessels.



A. *B.*

Fig. 4.—Thrombophlebitis of the deep veins of left leg in A.C., 32 years old. Venography performed before (*A*) and one month after the perivenous sympathectomy of the femoral and saphenous veins (*B*). In the second venography (*B*) there is marked increase in collateral venous circulation.

Does the efferent vasomotor innervation of the veins exert a similar influence on the development of the collateral venous circulation? In approaching this problem I employed three different methods of interrupting the venous sympathetic innervation in patients with phlebitis or thrombophlebitis of the lower limbs.

Series 1.—In four patients, two with acute phlebitis and two with thrombophlebitis of the lower limbs, repeated anesthetic blocks of the lumbar sympathetic chain (second, third, and fourth lumbar nerves) were performed. In these

cases the number of anesthetic blocks varied between four and thirteen; they were made over a period of ten to thirty days. Venographic studies with uroselectan were made before and at the end of the treatment. They showed that the repeated anesthetic blocks of the lumbar sympathetic chain not only relieved the pain and modified the vasomotor disturbances but the collateral circulation of the veins was increased (Fig. 3).

A. *B.*

Fig. 5.—Thrombophlebitis of the deep veins of the left leg in E.C., 47 years old. Venography performed before (*A*) and one month after the left lumbar sympathectomy (third and fourth lumbar nerves) (*B*). In the second venography (*B*) there is an increase in the collateral venous circulation in the lower third of the leg.

Series 2.—In two patients with thrombophlebitis of the veins of the legs, perivenous sympathectomy of the femoral vein was performed. Below the crural arcade the femoral vein was approached and the perivenous innervation was interrupted for a length of two inches. The innervation was also interrupted in the terminus of the saphenous vein. The operation was followed by relief from pain and amelioration of the vasomotor disturbances. The veins of the legs in these two patients were studied before and one month after the operation; visualization by x-rays showed a definite increase in the collateral venous circulation (Fig. 4).

Series 3.—Finally, in two patients with thrombophlebitis of the veins of the lower limbs the lumbar sympathetic chain was interrupted, the third and fourth sympathetic ganglia and the intermediate trunk being resected. A manifest improvement followed the operation in each case. Venographic studies one month later showed in each a definite increase in the venous collateral circulation (Fig. 5).

In these three series of patients with phlebitis or thrombophlebitis, interruption of the efferent vasomotor innervation of the veins of the lower extremities by repeated anesthetic blocks of the lumbar sympathetic chain, perivenous sympathectomy, or lumbar ganglionic (third and fourth lumbar nerves) sympathectomy was followed by an evident increase in the venous collateral circulation.

SUMMARY AND CONCLUSIONS

1. The veins are innervated by afferent sensory and efferent vasomotor pathways. Direct mechanical or chemical stimulation of the afferent pathways at the level of the wall of the veins may cause pain. The relief of venous pain and venospasm in acute phlebitis and thrombophlebitis by intravenous anesthesia of the inflamed vein or by the anesthetic block of the sympathetic chain, for a period longer than the anesthetic action of the drug employed, suggests that venospasm may play an important role in the mechanism of pain.

2. The observations in these cases demonstrate also that acute phlebitis or thrombophlebitis may be accompanied by venospasm extending far beyond the phlebitic or thrombosed vein. This venospasm may be relieved by peripheral anesthesia of the venous wall or by interruption of the efferent pathways of the sympathetic chain.

3. In cases of thrombophlebitis it was observed that repeated anesthetic blocks of the sympathetic chain with procaine hydrochloride, perivenous sympathectomy, or resection of the regional sympathetic chain was followed by an increase in the collateral venous circulation.

4. These investigations have demonstrated that physiologic or anatomic interruption of the innervation of the veins may relieve the pain and the venospasm, and it also increases the development of collateral circulation.

5. The facts observed suggest that the plan of the innervation of the veins in relation to pain, spasm and collateral circulation is similar to that of the afferent sensory and the efferent vasomotor pathways of the arteries.

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THE PRACTICE OF SURGERY IN JAPANESE PRISON CAMP HOSPITALS IN BURMA AND SIAM

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IN THIS paper an attempt will be made to record briefly the diagnostic and therapeutic methods adopted by the surgical staff in the primitive so-called hospitals of the Japanese Prison Camps in 1942-1945.

I was the senior medical officer and surgeon in charge of 1,800 United Kingdom, Australian, Dutch, and American prisoner of war patients at Kohn Kuhn, an abandoned working camp, Burma, in 1943, and chief medical officer and consulting surgeon at the prisoner of war hospital camp of 8,000 patients at Nakampaton, Siam, in 1944-1945. The Japanese medical officer confined his activities to discharging men for work. The Burma-Siam Railway was under construction and the Japanese were forcing the pace. Every prisoner who could work or even walk was compelled to do his utmost. The diet was meager—rice in sufficient quantities and polished, but meat supplied in amounts varying from 1 to 2 ounces per man per day. Vegetables were of the cucumber-radish-spinach variety. Protein deficiency was marked.

Pellagra.—Pellagra was responsible for many deaths.

Patients were admitted to the jungle camp hospital in all stages of debility. Sore mouth, fissured tongue, sore pharynx, anorexia, and diarrhea were common. Skin rashes of the pellagrous type were seen in a few. Mental changes occurred in a small number. Post-mortem examination revealed in all the shrunken organs, spleen, liver, and heart, and a "diaphanous" bowel, a small, thin-walled stomach and intestine, so-called "rabbit gut," and in some a shrunken brain with secondary hydrocephalus and convolutional atrophy.

Malaria was universal and quinine, the only drug supplied by the Japanese, was provided for 300 patients, that is, those patients admitted with malaria as the primary disease. One disease was permitted by the Japanese Medical Service, hence the patients with pellagra, dysentery, and tropical ulcer were "not allowed to have malaria" by order of the Japanese.

No treatment was available for the patients with pellagra. Food was insufficient and nicotinic acid, even if available, would not have cured them, as the changes in many were obviously irreversible.

Dysentery was not the scourge it was in some camps. Hygiene was reasonably good. A hut of 100 was labeled "the death house" and to it were taken patients with infectious diseases (bacillary and amebic). Some died following perforation of the colon; others developed masses, especially in the right illiac fossa; and still others sloughed great casts of the colon and rectum before they died. My death reports, which the Japanese always insisted should be written in detail, were condemned by the Japanese doctor at headquarters because I used the term amebic dysentery. It was forbidden; the word diarrhea had to be substituted. After protests and much correspondence, followed by a

visit from a Japanese Lieutenant Colonel, it was admitted that a clinical diagnosis of amebic dysentery in the absence of microscopic findings would be accepted and apologies were offered that there was no Japanese supply of emetine. A colleague procured for me in another Japanese camp, a bottle of ipecacuanha extract from which a Dutch officer, who acted as one of my medical orderlies, extracted 150 1 gr. doses of emetine. A short course of five injections of this homemade alkaloid was given to fifty patients with wonderful results.

I, personally, having suffered from amebic dysentery can speak feelingly of the dramatic effect of emetine. Many of the patients had ulcers in the rectum, and a proctoscope made from tin was constructed which enabled me to obtain a good view of the whole rectum when the patient was placed in the knee-elbow position. Sunlight reflected by a mirror illuminated the interior of the rectum and much information concerning the progress of the lesions was gained from regular rectal inspections. Ulceration and necrosis of the "valves" was common and in some was followed by stricture. Dilatation by bamboo dilators was practiced and succeeded in most; colostomy was required in one patient because of obstruction. This patient was later, at Nakompaton, subjected to a Kraske type of resection of the local fibrosed rectum with excellent results.

Ulcer.—Ulcer of the leg and occasionally of the hand, arm, and body occurred in great numbers. In that camp 500 cases were classified as tropical ulcer. At least sixty patients had ulcers on both legs, some had two or more ulcers on one leg. The term tropical ulcer was used for this condition, but as my Dutch colleague pointed out, the ulcers did not conform to the type commonly seen in the tropics and known by that name.

Until May, 1943, I had treated some hundreds of ulcers of the leg in prisoners of war in Sumatra and Burma. They were the usual circular ulcer from 1 to 5 cm. in width, circular, having a sloughing base on the fascia.

Foments, curettage, and iodoform and bandaging for several days were usually sufficient to bring about healing. There were many in the 500 previously mentioned which were of the usual type, but many followed a more malignant course. Spreading peripherally, eroding the fascia, periosteum, and even blood vessels, they enveloped the leg, leaving muscle and bone exposed. Black necrotic areas of fascias and tendons were excised. An attack of malaria or an exacerbation of pellagrous diarrhea was accompanied by a rapid spread of the ulcer. The usual methods of curettage, foments, etc., failed. Iodoform was not available. Where the process involved tendons and muscle, radical excision of those structures was carried out—even the tendo achillis were excised in a number of patients, the tibialis anterior, posterior, and the peroneal tendons were often cut out. Altogether sixty patients were treated in that fashion. Many recovered, the leg was saved, and surprisingly little loss of function resulted. Despite these measures, and all attempts at conservatism, the process in many spread up the periosteum of the tibia, opened the ankle joint, or caused such widespread gangrene of muscles that amputation was done. The mid-thigh

amputation was performed in most; twenty-five amputations were done below the knee.

Fortunately, I had a bottle of cocaine tablets in my possession and as no general anesthetic or novocain was available I experimented with spinal cocaine. One and one-half cubic centimeters of a 2 per cent solution sufficed to produce anesthesia to the groin and in most cases did not paralyze, so that the patient cooperated with the surgeons in the operation.

In that camp 120 legs were removed. All patients survived operations but many died months later of pellagra, malaria, or dysentery. The survivors number 50 per cent but they slowly made progress and are among the most grateful patients one could ever have.

Many problems confronted us in the camp. No facilities for operating were provided or, indeed, allowed. The abandoned bamboo huts of the former working camp provided the accommodation and a small annexe to a hut, open to the four winds of heaven on three sides, was the operating theater. A bamboo table was set up; towels and odds and ends of clothing were boiled up as dressings. No antiseptics were available, but we overcame this difficulty by using homemade rice alcohol. This was prepared in 90 per cent strength from the scraps of rice thrown away by the pellagrous patients. There was plenty of it. A small quantity of soap was obtained from the Japanese and this was supplemented by soap made locally from scraps of fat and ashes.

Catgut was necessary as I was afraid to use nonabsorbable ligatures for the femoral artery, in the presence of so much sepsis. Cotton was used for skin suture.

The catgut was prepared under my instructions from the peritoneum of the small intestine of the local ox, killed for food (the submucous coat proved too fragile). Strips of peritoneum were twisted on a stick and my medical orderlies became expert at it. Sterilization of the catgut was done by placing it in a tin in the rice oven, after the fires were raked out. After this was repeated for sport killing the gut was stored in the alcohol mentioned previously.

A reliable ligature was thus made which was used not only for arteries but also for suturing the abdominal wall in a few patients who came to immediate operation. A better method of sterilizing gut was later devised, that is, suspending a tin of the wound gut in another tin of boiling saturated salt water for one hour at a time.

A few cases of secondary hemorrhage occurred at the stump and in view of this risk, in patients being transported to Siam when the camp was clearing, I ligatured the femoral artery in Scarpa's Triangle as well. Amputation was circular with short flaps sutured anteroposteriorly in some—in others, loosely held together by one or two sutures because of sepsis.

The absence of pus formation was an outstanding feature in the cases mentioned. Toward the end of 1943 I organized an underground supply of meat to the sick, paying recovered patients of the more daring type to bring in cattle from the neighboring villages to the precincts of the camp and killing

at night. This was done in defiance of the Japs and under the nose of the guards. The effect of the extra meat was soon obvious: large ulcers began to suppurate, exposed bone became a sequestrum, and the limb was saved. Some of these patients were later operated on, the sequestrum in some cases being three-quarters the length of the tibia and the complete anterior half of the tibia being successfully removed. A single one sustained a fracture in the remaining involucrum, but union occurred in time.

In my daily visits to these patients with severe ulcers (I could see only seventy or eighty in a morning) how I welcomed a little pus and how encouraged the patient was when the dreadful pains of necrosis and ischemia gave way to inflammation with its local sequelae.

No microscopic examination of the discharge, which was usually thin and watery, was possible. In a few cases the muscles in the amputated limb were brick red in color suggesting an anerobe, but there was only one case of fulminating infection simulating gas gangrene and that in a medical orderly in the ulcer hut, who scratched the back of his hand on the bamboo bed of a patient with ulcer. Amputation of the right arm was necessary. The usual history was of a scratch by bamboo or stone, the abrasion failing to heal and spreading as an ulcer. The edges were necrotic with a tendency to undermining. The base was a slough, sometimes black and adherent, at other times soft and exuberant.

Nutritional Edema.—Nutritional edema was common, often called beriberi, especially for the records of the Japanese who actually understood that term.

Ascites, pleural effusions, edema, of limbs, and finally fatal edema of lungs and glottis occurred in some. Should the patient survive the edematous stage the fluid usually drained away and he died an emaciated skeleton. Meat and restricted fluids were the treatment, but only in the later stages at this camp was this feasible, and then many were past help.

Limbless men were provided with a light bamboo pylon and crutches (camp made). About 300 patients died in this camp in six months. The residual sick, together with similar patients from camps elsewhere along the railway, were transported by train to a newly constructed sick depot at Nakompaton, 45 kilometers from Bangkok.

SUMMARY

As chief medical officer and consulting surgeon at this "hospital" which accommodated 8,000 sick prisoners of war at one time I continued to work from January, 1944, to August, 1945. The majority of the cases were medical, for example, 1,500 chronic dysentery (mostly amebic), 1,100 avitaminosis, 1,200 surgical, the remainder skin, permanent disabilities such as amputations, and convalescents. The conditions in this camp were better than anything we had experienced in the jungle, but were far below the standards of most primitive European hospitals. There were no beds or bedding, and no specific drugs except quinine. Improvisation of instruments and operating equipment continued on a large scale, but the arrival of American Red Cross stores including some emetine and the sulfa drugs in July, 1944, relieved the situation. This

supply formed the basis of our dispensary stock; surgical instruments so supplied were retained by the Japanese. Operating was done with naked hands, scrubbing up in dishes with soap, and then immersion in camp-made 90 per cent alcohol. British, Dutch, and Australian surgeons worked as a team. A concrete floor and fly wire were provided in a special hut for the operating theater. Great attention was paid to detail of hemostatis and antisepsis (each layer in a hernia or appendix operation was painted with alcohol before closing). In 120 patients with herniotomy only two developed a mild sepsis. The soldering iron was used as cautery in mouth, neck, rectal, and pelvic operations. Silver clips made from beaten forks and fine cotton were freely used as hemostats. Homemade catgut was used for abdominal suture and ligature of large vessels. Nearly 1,000 major operations were performed. Patients with peptic ulcer were regularly "milked" for acid which was used for patients suffering from hypochlorhydria.

Spinal anesthesia* and local anesthesia were employed almost exclusively.

Fifteen hundred transfusions of defibrinated blood were given to debilitated and anemic patients. Cross typing was carried out in the usual way.

Sequestrectomy and skin grafting of old ulcers was done extensively.

Patients with chronic colon infection despaired of by the physicians were submitted to ileostomy. Physiotherapy and rehabilitation were important activities in 1944-1945. One hundred seventy-eight limbless men were fitted with camp-made wooden artificial limbs.

Full details of the surgical work have been recorded in the Official War Diary of the camp, copies of which have been supplied to the medical directors of the respective nations.

Naturally, in the absence of x-ray and other diagnostic equipment, surgeons were forced to rely on the case history and the physical findings. The eyes, fingers, and other senses were excercised more freely. Such an enforced return to fundamental methods was not without compensation. Analyses of post-mortem results and discussions at "death" meetings had a salutary effect and indicated that diagnosis was not so frequently in error as might be imagined.

In March, 1945, the Japanese prohibited post-mortem examinations for obvious reasons.

The technical work in the surgical field was of a good standard and great credit is due to the surgeons, Lieutenant Colonel Dunlop (A.A.M.C.), Major Kranz (A.A.M.C.), Captain Maconachie (R.A.M.C.), Captain Winckel (D.A.M.C.); to Captain Marcowicz (R.A.M.C., blood transfusion officer and biochemist), Captain Cyril Vardy (R.A.M.C., my adjutant and registrar), Major T. Marsden (R.A.M.C., pathologist). Doctors Chapman and Kostermanns, scientists; and to my colleagues of the Triumvirate of Control, Lieutenant Colonel Malcolm (O.B.E., M.C., R.A.M.C.), and Lieutenant Colonel Larsen (D.A.M.C.), and to the many medical orderlies of four nationalities who contributed so much to the success of the surgical department of the Nakompaton Prisoner of War Hospital.

*Cutocaine, American Red Cross.

CEREBRAL FUNGUS FOLLOWING PENETRATING WOUNDS

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A REVIEW of 426 cases of penetrating wounds of the head personally studied in two neurosurgical centers revealed 32 instances of cerebral fungus. The incidence was thus 7.5 per cent. All of the patients were battle casualties, having been injured in the Mediterranean Theater of Operations during the period of November, 1942, to February, 1945. As in the vast majority of that 20 per cent of this particular series of brain wounds in which deep infections developed, the organisms were of relatively low virulence.[1]

Cerebral fungus is a subacute or chronic herniation of the brain through a defect in dura, skull, and scalp, accompanied by infection, deep or superficial, or both. In chronic infected hernias progressive organization of the extruded and superficial portions into scar tissue is usually evident. The exact time of transition from cerebral herniation (the acute phase) to that in which a fungus may be said to exist depends, in our definition, upon these tissue changes. Gross evidence of such processes characteristic of fungus has usually begun to appear at the end of seventy-two hours. Cerebral fungus formation is one of the more serious complications of any craniotomy, but it is by no means a hopeless or necessarily a fatal one.[2-7] It is regarded as the result of failure of wound healing in the presence of infection; thus, while the fungus per se may require excision, attention must be directed primarily toward the diagnosis and elimination of such underlying causative factors as cerebral abscess or hematoma.

While the severity of the infection, the extent of tissue loss or damage, the size of the bone-dura aperture, and the state of the fungus were different in each patient, study of their causes and characteristics permits classification into two fairly distinct groups. In the first are included those cases in which the cerebritis was only superficial; bone or shell fragments might or might not have been retained. Intracranial pressure was normal or but moderately elevated. These we have termed "benign" fungi. Into the second group fell those cases in which local cerebritis, similar to that mentioned, was associated with, and increased by, cerebral abscess, hematoma, or a mass of necrotic brain tissue. In patients with abscess formation, retained bone chips or metallic foreign bodies could usually be demonstrated roentgenographically. There was always an increase in the intracranial pressure which was often progressive, and the growth of the fungus was relatively rapid. This type we have called "malignant" fungus.

BENIGN FUNGUS

Of this group of thirty-two patients with cerebral fungi, thirteen fell into the benign category. In each there was a superficial wound infection in the presence of an open dura mater. The protrusions grew slowly, they were not

shaggy nor sloughing, and they did not bleed easily. There were no symptoms suggestive of a high intracranial pressure, and neurologic evidence of extension of the process deep within the cerebrum was lacking. Retained bone chips were demonstrated in but four patients. These were removed at subsequent operation in three instances; neither abscess nor hematoma was found. The fourth patient was evacuated without operation because of the urgency of the military situation. A fifth individual was operated upon primarily for the excision of a fungus the size of a golf ball. This was found to be undergoing fibrosis and organization, and in retrospect it appeared likely that healing would have followed just as well had no operation been performed. Systemic but not local sulfonamide and/or penicillin therapy was practiced routinely in all cases. No patients were treated routinely by lumbar puncture; all hernias receded with eventual disappearance of the superficial infection, and with closure either spontaneously or surgically of the scalp defect. There were no deaths in this group.

A typical example of a benign fungus and its course is seen in Case 1.

CASE 1.—A soldier sustained a 4 cm. penetrating shell fragment wound of the vertex, to the left of the midline, in action in the Salerno landing, Sept. 9, 1943. There was a compound, comminuted, depressed fracture with a few indriven bone fragments. Right hemiparesis with motor aphasia was present. He was operated upon at an evacuation hospital twenty-four hours after injury, at which time scalp, bone, and cerebral portions of the wound were débrided with the removal of an estimated 7 Gm. of pulped brain. The dura was sutured, and the tripod incision of the scalp was closed without drainage. On Sept. 21, 1943, at a general hospital, the wound was noted to have become infected. By Sept. 25, 1943, a separation was present centrally with a small fungus presenting. A few small bone fragments were demonstrated by roentgenograms to be present in the extracerebral portion of the wound. Since there was no evidence of abscess or of sharply increased intracranial pressure, and since the hemiparesis was not increasing, no operation was undertaken. As the local infection cleared, the fungus receded and healing occurred satisfactorily. At no time while under observation was he critically ill, and he returned to the Zone of the Interior in good condition.

The forerunner of the fungus in this instance was the breakdown of the wound, superficial infection, and local cerebritis, together with a temporarily and moderately increased intracranial pressure. The latter slowly subsided with healing of the wound, thus making this fungus a self-limited complication.

Two patients entered the hospital six days after having been injured, Sept. 9, 1943, in the Salerno landing. Neither had been operated upon and the original dressings were still in place. Both patients had fungus formation, and the following brief record of one of them is illustrative of *this particular type*[4] of benign fungus:

CASE 2.—The patient was injured by shell fragments which guttered the left posterior parietal area, leaving a bone defect 2.5 by 3.5 cm. Many bone fragments were deeply indriven, and a few small metallic foreign bodies remained in the superficial layers of the brain. On arrival at the Head Center six days later there was a solid fungus filling the scalp defect, approximately 2.5 cm. in diameter. It was clean, firm, pulsating, and not hemorrhagic. There was no cerebrospinal fluid leak. A complete right homonymous hemianopsia was present. The patient was dull, passive, and aphasic to a marked degree in several spheres, but had no motor weakness. There was papilledema of 3 diopters. At operation on the same day the

scalp edges were excised, the bone edges freshened, and the tract débrided and cleansed of bone chips, hair, dirt, pulped brain, and a walnut-sized clot which extended down to the lateral ventricular wall. A solid scalp closure without drainage was effected. The patient made an uneventful recovery, having been left with moderate aphasia and right homonymous hemianopsia. He was sent to the Zone of the Interior shortly afterward with the wound well healed and the flap soft and pulsating.

This was a type of benign fungus occurring in an open wound which was not yet actively infected, but potentially so; there were retained bone chips and foreign bodies and an increase of intracranial tension due to the acute brain injury and clot rather than infection. Excision of the fungus, delayed but complete débridement,[8] and wound closure without drainage resulted in primary wound healing.

MALIGNANT FUNGUS

In this group are included those nineteen cases in which infected cerebral herniations, occurring subsequent to wound débridement, were associated with increased intracranial pressure caused by abscess, hematoma, or occasionally necrotic but uninfected brain tissue. Here the danger of meningitis and the complications of increased intracranial pressure were very real. Six of these patients died while under observation, and three others may have died later, since the military situation required their evacuation while critically ill.

The fungi, once started on their way through the open dura mater and bone, grew rapidly, as if propelled by the increased intracranial pressure behind them. As a rule they were shaggy, wet, and had a tendency to bleed. Signs and symptoms of increased intracranial tension were usually evident. While unfortunately the underlying nidus of infection, abscess, or clot can by no means always be located with exactitude by clinical signs, a roentgenologic demonstration of retained intracerebral foreign bodies or of bone chips offers a strong, although not invariably reliable, clue to its location. Gas or a foreign body which can be demonstrated to have shifted positions in serial roentgenograms clearly indicates a fluid-filled cavity. A purulent sinus may lead through the fungus, along a missile or bone chip tract, to the abscess and thus be a valuable guide at operation.

Until the infected mass is evacuated, be it a frank abscess or a deep cerebritis with or without hematoma, increasing tension will not be brought under control, nor the progress of the fungus arrested. Systemic chemotherapy with sulfonamides and/or penicillin was employed routinely, but served as an adjuvant to, rather than a substitute for, surgery. Operative removal of the causative factor is therefore urgently required in every case of malignant cerebral fungus. Delay or failure to find the associated abscess was responsible for the fatal outcomes in all of those patients who died. Meningitis may follow rupture of the abscess into the ventricle, the ventricle may rupture externally through the fungus, or death may result from the effects of increasing intracranial pressure.

Such a tragedy is illustrated by Case 3, in which the abscess, although sought for, was not found until too late.

CASE 3.—A 23-year-old engineer was struck in the left parietal region by a shell fragment on Oct. 13, 1943; a compound, comminuted, fracture resulted, with numerous indriven bone fragments. Right hemiparesis with partial aphasia followed. The wound was débrided eight hours later at an evacuation hospital and a foreign body and most of the bone fragments removed. Eight days later the patient arrived in North Africa. Purulent drainage from the wound was noted. The neck was somewhat rigid and the cerebrospinal fluid cell count was 8,700. Under sulfadiazine he was said to have improved gradually and was actually out of bed walking about until Nov. 19, 1943, thirty-seven days after injury. At that time severe headache and vomiting set in, the temperature rose to 103° F., and cervical rigidity reappeared. On admission to the Head Center on that date he complained of severe frontal headache. Incomplete nominal aphasia and right hemiparesis were evident. The neck was stiff and the temperature remained elevated. The cerebrospinal fluid cell count was 1,150, of which 850 were polymorphonuclear leucocytes. In the center of the left parietal wound there was a tense bulging fungus partially covered with granulation tissue. It was not pulsating. Some purulent drainage was evident. Roentgenographic examination disclosed a few, tiny, scattered bone chips beneath the defect and a metallic foreign body in the posterosuperior portion of the left temporal lobe. A diagnosis of brain abscess associated with incomplete débridement was made. The following day, under local anesthesia, the hernia was excised flush with the dura. Four sinus tracts were found and followed to their depths. Several bone fragments were evacuated, but no definite abscess, hematoma, nor mass of necrotic tissue was encountered, although one of the tracts extended approximately 7 cm. down into the temporal lobe. The hernia itself was quite necrotic and the walls of the sinus tracts were fibrotic. Cultures from the wound subsequently revealed anerobic hemolytic streptococcus and aerobic nonhemolytic staphylococcus.

Although the brain was moderately slack at the close of this procedure, we were far from satisfied with the findings, or lack of them. Despite the fact that the spinal fluid pressure remained elevated (280 to 300 mm. water) one was lulled into a false sense of security by the improvement in the patient's general condition and the lessening of signs of meningeal irritation. Actually, by Nov. 23, 1943, the spinal fluid pressure had become normal (120 mm. water). However, on Nov. 27, 1943, he complained of more headache. Spinal fluid pressure was found to be 210 mm. For the first time evidence of right homonymous hemianopsia and astereognosis in the right hand were observed, and the right hemiparesis, which had decreased following the second operation, became more pronounced. Gradually the fungus recurred. Lumbar punctures, performed one to three times a day, revealed pressures of 240 to 280 mm. water with cell counts ranging from 74 to 147. On Nov. 30, 1943, while sitting up smoking, he suddenly lost consciousness, the pupils became dilated and fixed, and stertorous respiration set in. The fungus increased in size rapidly, with much necrotic brain tissue being extruded. In vain the temporal and parietal lobes were tapped with a ventricular needle. No abscess was encountered. Another attempt the following day was successful in reaching a thick-walled abscess cavity in the temporal lobe. However, the patient's course was progressively downward and he died on Dec. 3, 1943. Autopsy revealed a large, well-encapsulated abscess of the left temporal lobe extending into the hippocampal region. No bone fragment was found within the cavity nor in its walls. The metallic foreign body lay one-half inch posterior to the wall of the abscess and was nicely encapsulated.

The mistakes here were incomplete wound débridement initially, and second, failure to employ ventriculography when search for a diagnosed abscess was unsuccessful. If the associated abscess be evacuated relatively early, wound healing and recovery may proceed rapidly, as exemplified in Case 4.

CASE 4.—A 31-year-old infantryman was struck in the left frontal region by a rifle bullet on Oct. 16, 1943. The wound was débrided, the missile removed, and closure effected the following day at a field hospital. A few days later at another institution the wound was observed to have broken down and a fungus was evident. The amount of sulfonamide administered was unknown, and penicillin was not then available. On the patient's arrival

at the Head Center on Oct. 25, 1943, eight days after the initial operation, the wound was found to be draining pus and a tense, shaggy, nonpulsating fungus was present. Neurologic examination revealed mild right hemiparesis and early papilledema. Mental processes were slow, but he was rational, cooperative, and complaining of but slight headache. There was little fever, and the neck was moderately stiff. Roentgenograms revealed several retained bone fragments a short distance beneath the defect in the posterior portion of the left frontal bone.

Under novocain anesthesia the fungus was excised flush with the dura. A mass of necrotic tissue and bone fragments was found beneath, associated with a partially encapsulated abscess. Following evacuation of the latter, and secondary débridement of the wound, the scalp was closed loosely about a drain. Wound cultures revealed aerobic hemolytic *Staphylococcus albus,* and aerobic hemolytic streptococcus. The highest recorded cerebrospinal fluid pressure after operation was 200 mm. of water, with a cell count of 330, of which 40 per cent were polymorphonuclear leucocytes. Convalescence was uneventful and the neurologic recovery apparently complete.

The history of the next patient whose injury was very extensive, illustrates some of the difficulties which may be encountered in the treatment of malignant fungus, where the latter, of itself, is of quite secondary importance. Inadequate débridement, both at the first and at subsequent operations, was apparently responsible for the fatal outcome.

CASE 5.—A soldier was wounded in action in Italy, July 15, 1944, sustaining multiple shell fragment wounds of the left side of the face, left eye, both supraorbital regions, and the right shoulder. There were multiple fractures of both frontal bones, orbital plates, ethmoids and sphenoid, as well as the left temporal and maxillary bones. There was deep injury to both frontal lobes and to the left temporal and parietal lobes. A diffuse shower of coarse and fine bone and metallic foreign bodies extended from the face into the left occipital region. The first operation was performed at an evacuation hospital, where a left frontal craniotomy was done for removal of subdural and extradural clots; in addition, wounds of the face and scalp were débrided and the left eye removed. The large frontal dural opening could not be closed. A gauze pack was placed at the base and brought out through the orbit. The pack was said to have been removed within one week. The scalp incision healed and the patient's general condition improved greatly. However, by July 28 a fungus had filled the defect in the orbital wound through which the pack had been brought out, and a spinal fluid fistula made its appearance there as well.

On arrival at the Neurosurgical Center July 30, 1944, all wounds were healed save the orbit, where the fungus was wet and necrotic. The patient was mentally disturbed and showed many and varied neurologic defects indicative of bilaternal cerebral injury, together with signs of impending meningitis. On Aug. 2, 1944, a left temporal craniectomy was performed with removal of subdural and intracortical hematomas, and evacuation of an abscess (nonhemolytic *Staph. albus*). Bone chips and the large foreign body which lay free in a soft, necrotic, pathway leading into the posterior temporoparietal region were removed. This foreign body had been seen to shift position several times in serial films. The poor general condition of the patient prohibited redébridement of the frontal lobe wounds at that time. He improved markedly after this operation, but the fistula about the orbital fungus continued to flow, and because of the danger of meningitis developing he was again operated upon Aug. 8, 1944. On this occasion the fungus and the adjacent necrotic portion of frontal lobe tissue were removed and extensive fascia lata grafts were employed to repair the large dural defects presenting at the opened frontal and ethmoid sinuses. Cultures from the cerebrospinal fluid fistula showed *Bacillus aerogenes* and *Staph. albus.* He did well until Aug. 18, 1944, when his condition became suddenly and temporarily much worse and the scalp flap swelled out under marked tension. The left ventricle was tapped, the fluid cultured, and *Escherichia coli* found. Improvement was again rapid. Sept. 5, 1944, the patient once more suddenly became very ill, and again a ventricular tap yielded a turbid fluid, rich in

cells, with many bacilli and cocci on a stained smear. Sept. 6, 1944, the left temporoparietal lobe was re-explored but no abscess was found. Roentgenograms, meanwhile, had not indicated any shift in any of the remaining foreign bodies. He died on Sept. 9, 1944. Necropsy revealed a mass of seminecrotic tissue between the floor of the left ventricle and the snugly healed dural patch over the orbital roof.

In the light of the next case (Case 6), exploration of the frontal lobe with redébridement should have been done and might well have prevented the patient's death.

That a cerebral fungus associated with a rupture of the ventricle and a wide scalp defect need not invariably be fatal, is illustrated by Case 6.

CASE 6.—A German soldier, aged 19 years, was struck in the left supraorbital region by a .30 caliber bullet on Sept. 20, 1944. The wound was dressed with sulfonamide powder and on the same day he reached one of our evacuation hospitals where the wound was débrided. The large dural defect was repaired with a pericranial graft. The incision, because of loss of tissue, was said to have been closed under great tension. Penicillin was used both locally and intramuscularly (25,000 units injected into the cavity at the close of the operation and 15,000 units injected subsequently each day). Although his general condition improved, a wound infection developed, with colon-smelling pus. Sept. 26, 1944, the wound was opened widely, the exposed portion of the dural graft removed, and pus and necrotic tissue sucked away. A drain was left in the wound. By the following day the temperature had risen to 105° F. and the neck was stiff. Bacteriologic examination revealed "spore-forming anaerobes." Penicillin, 10,000 units, was injected intrathecally each day in addition to that given by routine intramuscular administration. The patient remained comatose until Sept. 29, 1944, at which time the temperature and pulse returned approximately to normal. He began to respond to questions and to take food.

He was then evacuated through two other hospitals, at the latter of which he was seen for the first time by one of us on Oct. 21, 1944. Apparently, he had been holding his own fairly well. Cervical rigidity had disappeared, but pus continued to drain from the wound. Two days previously, cerebrospinal fluid was observed to be issuing from the wound. He then became listless and the temperature rose to 103° F. When seen prior to transfer to the Head Center, the temperature was 104° F., the neck quite stiff, and a large cerebral fungus was present in the left frontal wound, from which there was a copious flow of cerebrospinal fluid. Roentgenographic examination revealed a bone defect 6 cm. in diameter with no retained bone fragments. Cultures from the wound and from the lumbar spinal fluid on at least two occasions disclosed the presence of *Esch. coli*. The cerebrospinal fluid cell count varied from 3,600 to 3,950 cells, with 66 to 72 per cent polymorphonuclear leucocytes.

On Oct. 22, 1944, immediately after admission, a craniotomy was performed under ether anesthesia. After the fungus had been excised, the fistula was seen to extend directly into the anterior horn of the left lateral ventricle. A radical débridement of the left frontal lobe was performed. The frontal sinus was apparently healthy and was not disturbed. The cavity was thoroughly washed with saline solution and 10,000 units of penicillin were instilled. In order to close the huge scalp defect a pedicle flap was brought down from above and the bed of the latter covered by a split-thickness graft placed on the remaining pericranium.

The postoperative course was marked by a temperature rise as high as 102.6° F. the following day, after which it returned to within one degree of normal. From time to time during the next two to three weeks there were episodes of fever and increased fluid under the flap, requiring aspiration. However, the scalp wound healed per primam and the graft took well.

Approximately one month later colon bacillus meningitis recurred. The flap bulged tensely and a re-exploration of the wound was deemed imperative. This was carried out by reopening the lower limb of the old incision. A large smooth-walled cavity was revealed, lined with glistening arachnoidal membrane beneath which numerous silver clips were visible.

No reaction was evident about any of them. The opening into the ventricle was 2 to 3 mm. in diameter; a small mass of seminecrotic tissue containing a few particles of dirt had ruptured through the membrane. This was thoroughly excised back to normal-appearing tissue. The wound was closed as before without drainage. Convalescence from that time on was quite satisfactory, and the patient, when last examined four months after the last operation, was ambulant, without complaints, and with no gross neurologic disturbances.

Cerebral fungi occurred in approximately one-quarter of all deep and superficial infections. The mortality was definitely higher in those deep infections in which fungus developed. They appeared less frequently in those wounds in which the dura had been closed. Hence, dural closure by suture or by living graft is desirable at the time of wound débridement. An additional reason for carrying out this procedure is that the sutured or patched dura may shield the brain effectively from a superficial infection. This has been observed by one of us on at least eight occasions in brain and spinal cord wounds.

SUMMARY

1. Thirty-two cases of cerebral fungus which occurred among 426 penetrating skull wounds were studied.

2. These were roughly divisible into two groups: the first, termed "benign," consisted of locally infected herniations associated with superficial wound infections; the second, called "malignant," occurred in conjunction with deeply infected wounds, overlooked hematomas, or pockets of necrotic tissue. In the majority of these the dura had been left unsutured, although in a few it had been forced open by pressure and infection.

3. All save one of the thirteen "benign" fungi were small, grew slowly, and were not associated with progressive neurologic changes. Intracranial pressure was elevated but moderately, if at all. The natural tendency was to heal within a few weeks. Only one, the largest, was excised, and it appeared to have been undergoing fibrosis. Systemic chemotherapy was practiced routinely. There were no deaths in this group.

4. Among the nineteen cases of "malignant" fungus, there were six known deaths and possibly three others subsequent to evacuation. These hernias were large and shaggy with a tendency to bleed. Continued growth of the mass, mounting intracranial pressure, and progression of neurologic changes were characteristic of this group. Treatment entailed evacuation of the abscess or hematoma and/or redébridement of the wound supplemented by systemic sulfonamide and/or penicillin therapy. Excision of the fungus flush with the dura was thus not the essential part of the operation, although it served to lessen pressure in the region of the dural opening[9, 10] and to afford access to the principal source of trouble. In each of the six known fatalities an underlying abscess was overlooked or evacuated too late. With few exceptions these patients can be saved by proper and timely surgical intervention. Systemic chemotherapy with penicillin and/or sulfonamides is apparently of value in preventing meningitis. Should signs of meningeal irritation or invasion appear, penicillin should be employed intrathecally as well, and every effort directed toward elimination of the source of infection. To do less is to court imminent disaster.

5. Case histories exemplifying both successful and unsuccessful treatment are cited, and what are believed to have been the errors committed pointed out.

6. Dural closure, in addition to painstakingly thorough wound débridement at the time of the initial operation, is recommended.

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Book Reviews

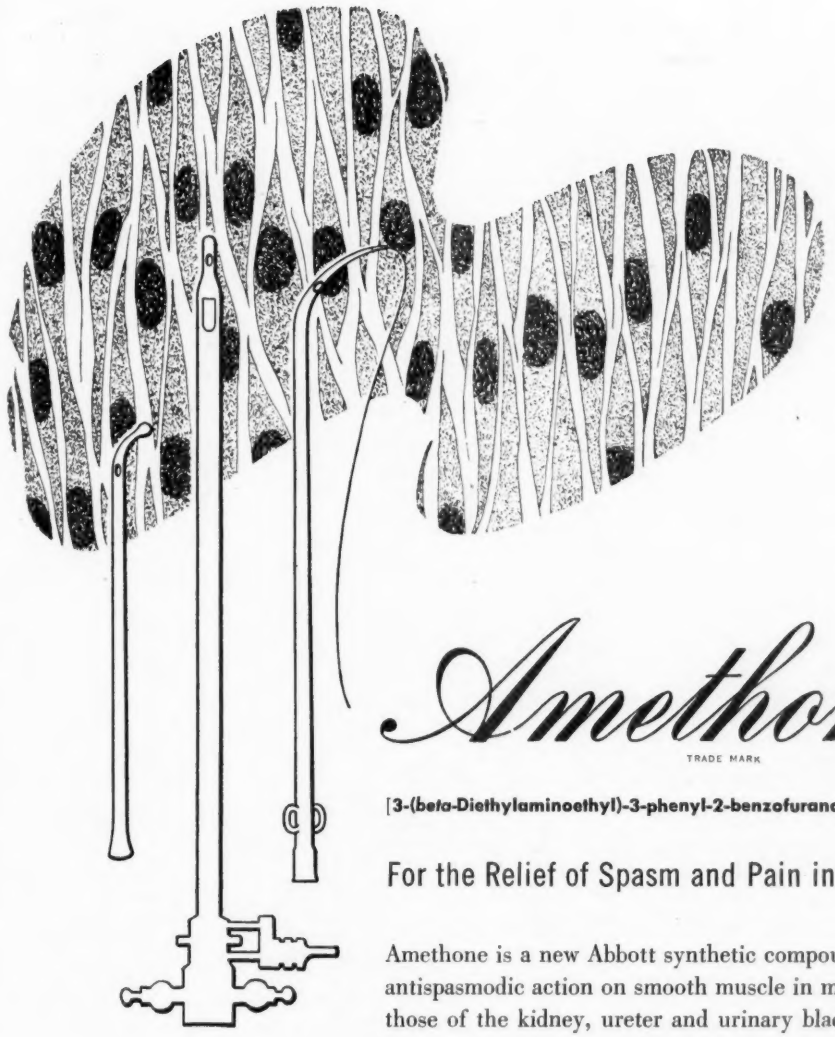
Modern Urology for Nurses. By S. M. Dwyer, R.N., B.S., and G. W. Fish, M.D. Ed. 2. Cloth. Pp. 287, with 66 engravings. Philadelphia, 1945, Lea & Febiger. $3.25.

This is a compact volume intended to give the student and graduate nurse the fundamental principles of urologic nursing care. The various problems encountered are well covered and the anatomic, physiologic, and pathologic features of conditions requiring care are discussed so that the nurse should have a comprehensive concept of the underlying disease. Nursing care has then a rational basis and, with understanding, care improves and becomes enthusiastic rather than indifferent.

The chapter on establishment of an urologic section emphasizes the importance of segregation of patients with urologic disorders so that trained staff and special necessary equipment may be available. Throughout the book the necessity of understanding in the treatment of the sensitive, embarrassed patient is stressed and the discussion of the nurses' role in reassurance and teaching of the patient is laudable.

The authors are not dogmatic, but rather give fundamental grounding in urologic nursing care, to be modified by the particular routine of the urologists of any hospital. A description of the necessary staff and required equipment of the Squier Urological Clinic is given in some detail.

The chapter on teaching of urologic nursing seems particularly good. The brief discussions of operative procedures give understanding to the floor nurses and a foundation, to which the urologist should add, to the cystoscopic and surgical nurses.



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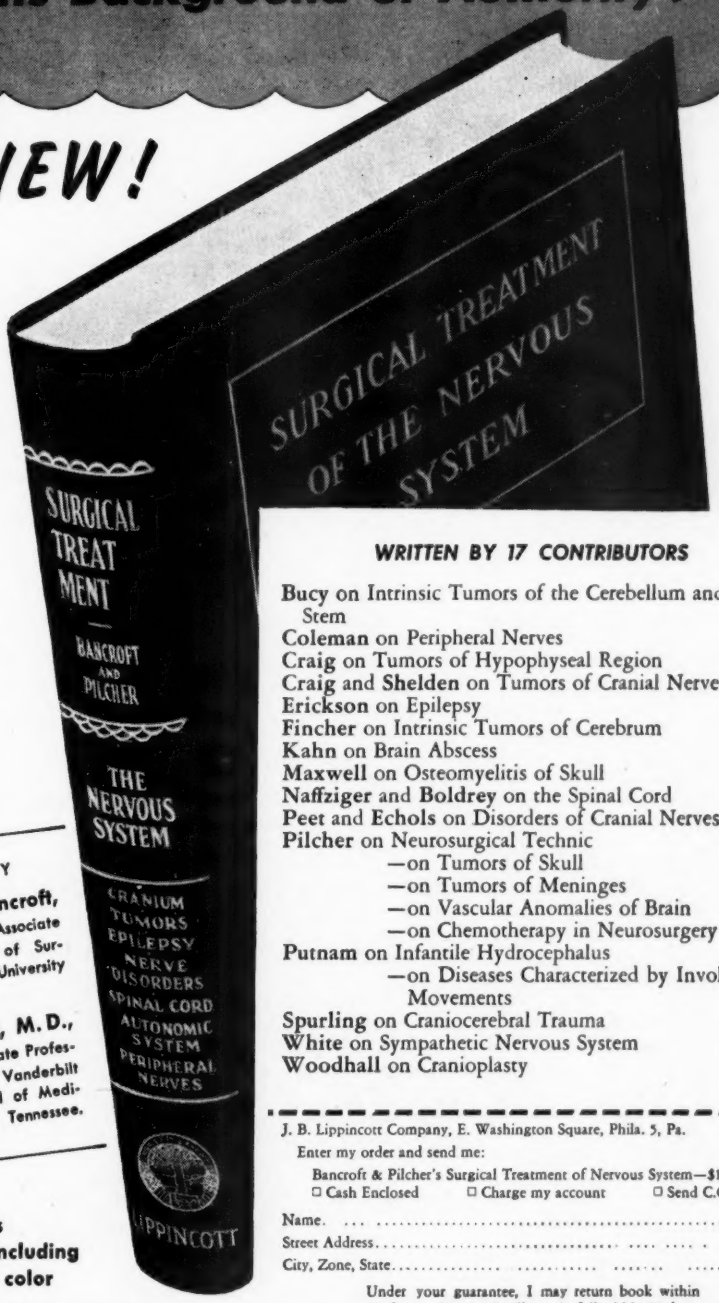

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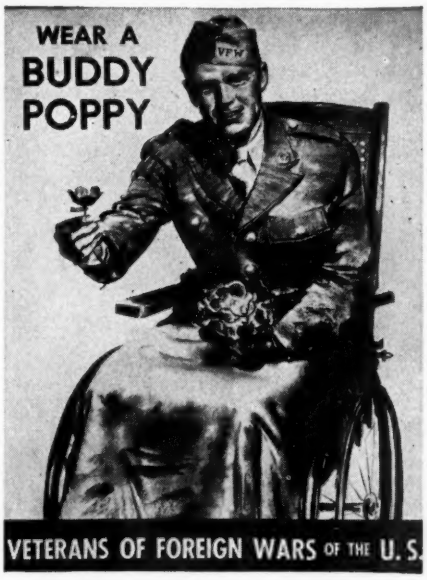

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My dear Commander:

The increasing importance of veterans' affairs particularly commends the V. F. W. sale of Buddy Poppies this year. In supporting the V. F. W. National Home at Eaton Rapids, Michigan, this poppy sale gives the orphaned children of veterans an opportunity in life equal to that enjoyed by the average American child. These are both splendid purposes which recommend to all the wearing of a Buddy Poppy this year.

Very sincerely yours,

Harry Truman

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One day every year is set aside to remind us that those who go to war are sometimes called upon to give something more than even life itself in defense of their country, and that there are many for whom the war and its suffering is not yet over.

The Veterans of Foreign Wars, the "Gold Stripe" organization, all of whose members have seen service overseas or in hostile waters, annually conduct a nation-wide sale of "Buddy Poppies" prior to Memorial Day as a means of perpetuating the memory of America's honored dead, by a practical and patriotic program of relief and service.

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